

THE BRITISH
JOURNAL OF SURGERY

THE BRITISH JOURNAL OF SURGERY

UNDER THE DIRECTION OF THE FOLLOWING
EDITORIAL COMMITTEE:

LORD MOYNIHAN, K.C.M.G., C.B. (Leeds), *Chairman*

H. T. DYKE ACLAND, C.M.G., C.B.E. (Christ-
church, N.Z.)

EDWARD ARCHIBALD (Montreal)

SEYMOUR BARLING, C.M.G. (Birmingham)

L. R. BRAITHWAITE (Leeds)

ARTHUR H. BURGESS (Manchester)

SIR F. POWELL CONNOR, D.S.O., Lt.-Col. I.M.S.
(Calcutta)

ARTHUR COOKE (Cambridge)

R. C. ELMSLIE, O.B.E. (London)

CHARLES H. FAGGE (London)

C. H. S. FRANKAU, C.B.E., D.S.O. (London)

JOHN FRASER, M.C. (Edinburgh)

ANDREW FULLERTON, C.B., C.M.G. (Belfast)

GEORGE E. GASK, C.M.G., D.S.O. (London)

W. SAMPSON HANDLEY (London)

SIR ROBERT JONES, Bt., K.B.E., C.B. (Liver-
pool)

ROBERT E. KELLY, C.B. (Liverpool)

HUGH LETT, C.B.E. (London)

SIR J. LYNN-THOMAS, K.B.E., C.B., C.M.G.
(Cardiff)

SIR ALEX. MACCORMICK, K.C.M.G. (Sydney)

SIR GEORGE H. MAKINS, G.C.M.G., C.B.
LL.D. (London)

WM. ERNEST MILES (London)

A. B. MITCHELL, O.B.E. (Belfast)

HENRY A. MOFFAT, D.S.O. (Capetown)

SIR THOMAS NYLES, C.B. (Dublin)

CHARLES A. PANNETT (London)

HARRY PLATT (Manchester)

SIR D'ARCY POWER, K.B.E. (London)

SIR PERCY SARGENT, C.M.G., D.S.O. (London)

GRAHAM S. SIMPSON (Sheffield)

WILFRED TROTTER (London)

G. GREY TURNER (Newcastle-on-Tyne)

SIR CUTHBERT S. WALLACE, K.C.M.G., C.B.
(London)

SIR W. I. de COURCY WHEELER (Dublin)

D. P. D. WILKIE, O.B.E. (Edinburgh)

ARCHIBALD YOUNG (Glasgow)

E. K. MARTIN (London), *Editor, Atlas of Pathological Anatomy*

E. W. HEY GROVES (Bristol), *Editorial Secretary*

VOLUME XVIII.

July 1930 to April 1931. Numbers 69 to 72.

Together with the Atlas of Pathological Anatomy, Numbers 21 to 24.

BRISTOL: JOHN WRIGHT AND SONS LTD.

LONDON: STATIONERS' HALL COURT. SIMPKIN MARSHALL LTD.

TORONTO: THE MACMILLAN CO. OF CANADA LTD.

CALCUTTA: BUTTERWORTH AND CO. (INDIA) LTD.; THACKER, SPINK AND CO. BOMBAY: W. THACKER AND CO.
SYDNEY: ANGUS AND ROBERTSON LTD. NEW ZEALAND: WHITCOMBE AND TOMBS LTD.

UNITED STATES OF AMERICA: WILLIAM WOOD AND CO. NEW YORK, *Sole Agents.*

PRINTED IN ENGLAND
BY JOHN WRIGHT AND SONS LTD., BRISTOL.

CONTENTS OF VOLUME XVIII.

NUMBER 69.

	PAGE
SOME BYGONE OPERATIONS IN SURGERY:—	
Cutting for the Stone - - - - - <i>Sir D'Arey Power</i>	1
ENDOTHELIOMA OF THE CAUDA EQUINA. REPORT OF A CASE WITH REMOVAL OF TUMOUR AND RECOVERY	
<i>Cecil P. G. Wakeley, C. Worster-Drought, I. M. Allen, and W. E. Carnegie Dickson</i>	6
HERNIA IN AFRICANS - - - - - <i>W. K. Connell</i>	16
CYSTS OF THE LONG BONES OF THE HAND AND FOOT - - - <i>Harry Platt</i>	20
GAS GANGRENE IN CIVIL SURGERY - - - - - <i>Nils L. Eckhoff</i>	38
THE APPROACH TO THE HIP-JOINT: A CRITICAL REVIEW AND A SUGGESTED NEW ROUTE - - - - - <i>R. P. Osborne</i>	49
IS IT TRUE THAT THE VALVES IN A VEIN NECESSARILY BECOME INCOMPETENT WHEN THE VEIN DILATES? - - - - - <i>R. E. Kelly</i>	53
THE SPLINTING OF CASES OF TUBERCULOSIS OF THE HIP - <i>M. Forrester-Brown</i>	54
A BACTERIOLOGICAL STUDY OF FIFTY CASES OF CHOLECYSTECTOMY WITH SPECIAL REFERENCE TO ANAEROBIC INFECTIONS	
<i>G. Gordon-Taylor and Lionel E. H. Whitby</i>	78
CARCINOMA OF THE DUODENUM - - - - - <i>J. Ewart Schofield</i>	84
TUBULAR HERMAPHRODITE WITH TERATOMA OF THE INTERNAL GENITALIA	
<i>E. D'Arey McCrea</i>	91
RODENT ULCER AS THE SEQUEL TO PROLONGED X-RADIATION	
<i>Harold Edwards, E. ff. Creed, and H. A. Colwell</i>	96
VOLVULUS OF THE STOMACH - - - - - <i>James Buchanan</i>	99
ON SOME ASPECTS OF THE PATHOLOGY OF HYPERTROPHIC CHARCOT'S JOINTS	
<i>E. S. J. King</i>	113
A STUDY OF TUMOURS AND INFLAMMATIONS OF THE GASSERIAN GANGLION	
<i>F. A. R. Stammers</i>	125
INTESTINAL OBSTRUCTION COMPLICATING POSTERIOR GASTROJEJUNOSTOMY	
<i>George Armitage</i>	154
SHORT NOTES OF RARE OR OBSCURE CASES - - - - - <i>Various Writers</i>	162
REVIEWS AND NOTICES OF BOOKS - - - - -	175

NUMBER 70.

SOME BYGONE OPERATIONS IN SURGERY:—	
Cutting for the Stone (<i>continued</i>). Frère Jacques' Operation	
<i>Sir D'Arey Power</i>	185
THE PATHOLOGY OF CONGENITAL TORTICOLLIS - - - - - <i>D. Stewart Middleton</i>	188

	PAGE
TWO CASES OF HEMANGIOMA OF THE BLADDER - - - <i>Jas. B. Macalpine</i>	205
THE ANATOMICAL RESULT OF PERI-ARTERIAL SYMPATHECTOMY <i>D. M. Blair, D. Duff, and J. A. Bingham</i>	215
OSTEOPLASTIC CRANIOTOMY: A NEW TECHNIQUE FOR THE RESECTION AND TURNING DOWN OF BONE-FLAPS FROM THE SKULL (Introduction by <i>A. W. Sheen</i>) <i>Lambert Rogers</i>	221
PURPURA AS AN ACUTE ABDOMINAL EMERGENCY - - - <i>Hamilton Bailey</i>	234
THE MIXED TUMOURS OF THE SALIVARY GLANDS - - - <i>D. H. Patey</i>	241
STUDIES IN EXPERIMENTAL MITRAL OBSTRUCTION IN RELATION TO THE SURGICAL TREATMENT OF MITRAL STENOSIS - - - <i>W. C. Wilson</i>	259
SOME COMPLICATIONS OF HYDATID DISEASE - - - <i>Harold Dew</i>	275
THE TREATMENT OF INFECTED OPEN FRACTURES - <i>Ernest W. Hey Groves</i>	294
AN OPERATION FOR THE COMPLETE EXPOSURE OF THE ELBOW-JOINT <i>H. W. L. Molesworth</i>	303
HEMANGIOMA OF THE KIDNEY - - - <i>W. Arthur Mackey</i>	308
SARCOMA AND TRAUMA - - - <i>R. J. Willan</i>	321
FATAL CARDIAC FAILURE OCCURRING IN PERSONS WITH ANGULAR DEFORMITY OF THE SPINE - - - <i>Carey F. Coombs</i>	326
SHORT NOTES OF RARE OR OBSCURE CASES - - - <i>Various Writers</i>	329
REVIEWS AND NOTICES OF BOOKS - - - - -	341

NUMBER 71.

SOME BYGONE OPERATIONS IN SURGERY:—

Cutting for the Stone (<i>continued</i>). Cheselden's Operation <i>Sir D'Arcy Power</i>	353
THE INTERVERTEBRAL DISCS - - - <i>N. Ross Smith</i>	358
THE MECHANISM OF DEEP TENDERNESS IN GASTRIC AND DUODENAL ULCER <i>John Morley and E. W. Twining</i>	376
ACUTE OSTEOMYELITIS OF THE SPINE - - <i>The late H. W. Carson</i>	400
A CASE OF OSTEOITIS FIBROSA (WITH FORMATION OF HYALINE CARTILAGE) <i>E. D. Telford</i>	409
A CASE OF PRIMARY ADAMANTINOMA OF THE TIBIA <i>Alfreda H. Baker and L. M. Hawksley</i>	415
THE COLLOID ADENOCARCINOMATA OF THE BLADDER VAULT ARISING FROM THE EPITHELIUM OF THE URACHAL CANAL: WITH A CRITICAL SURVEY OF THE TUMOURS OF THE URACHUS - - - <i>R. Campbell Begg</i>	422
STUDIES ON HIGH INTESTINAL OBSTRUCTION. THE ADMINISTRATION OF SALINE AND OTHER SUBSTANCES BY ENTEROSTOMY BELOW THE SITE OF OBSTRUCTION <i>J. C. Armour, T. G. Brown, D. M. Dunlop, T. C. Mitchell, H. H. Searls, and C. P. Stewart</i>	467
VICARY'S PREDECESSORS - - - - - <i>G. E. Gask</i>	479

CONTENTS

vii

	PAGE
THE TREATMENT OF GLIOMATA AND PITUITARY TUMOURS WITH RADIUM <i>Sir Percy Sargent and Stanford Cade</i>	301
SHORT NOTES OF RARE OR OBSCURE CASES - - - <i>Various Writers</i>	321
REVIEWS AND NOTICES OF BOOKS - - - - -	332

NUMBER 72.

SOME BYGONE OPERATIONS IN SURGERY :—	
An Historical Lithotomy: Mr. Samuel Pepys - <i>Sir D'Arcy Power</i>	341
THE USE OF LOCAL ANÆSTHESIA IN THE TREATMENT OF FRACTURES, WITH CONCLUSIONS DRAWN FROM FIFTY CASES - - <i>John P. Hosford</i>	346
THE VASCULAR COMPLICATIONS OF CERVICAL RIB <i>E. D. Telford and John S. B. Stopford</i>	357
OSTEOMA OF THE FRONTAL SINUS: WITH PARTICULAR REFERENCE TO ITS INTRA- CRANIAL COMPLICATIONS, AND WITH THE REPORT OF A CASE <i>George Armitage</i>	365
TRANSPLANTATION OF AN EXTERNAL BILIARY FISTULOUS TRACT INTO THE DUODENUM <i>George E. Waugh</i>	381
UNUSUAL COMPLICATION ARISING DURING OPERATION FOR A LARGE HYDATID CYST OF THE LIVER - - - - <i>James A. Jenkins</i>	385
RENAL CALCULI WITH SQUAMOUS CARCINOMA IN A HYDRONEPHROTIC KIDNEY <i>L. Woodhouse Price and Arthur Jacobs</i>	390
CONCERNING THE PATHOLOGY OF TUMOURS OF TENDON-SHEATHS <i>E. S. J. King</i>	394
THE TREATMENT OF CEREBRAL TUMOURS WITH RADIUM - <i>J. Paterson Ross</i>	618
INTERARTICULAR SYNOVIAL FOLDS - - - - <i>J. C. Boylean Grant</i>	636
INTRAVENOUS LOCAL ANÆSTHESIA - - - - <i>John T. Morrison</i>	641
NON-PARASITIC CYSTS OF THE LIVER: THEIR CLINICAL AND PATHOLOGICAL ASPECTS <i>F. D. Ackman and Lawrence J. Rhea</i>	648
SHORT NOTES OF RARE OR OBSCURE CASES - - - <i>Various Writers</i>	655
REVIEWS AND NOTICES OF BOOKS - - - - -	674

GENERAL INDEX TO VOLUME XVIII.

SUPPLEMENT: ATLAS OF PATHOLOGICAL ANATOMY.

FASCICULUS VI:	{	DISEASES OF THE JOINTS.
	{	DISEASES OF THE THYROID GLAND.

THE BRITISH JOURNAL OF SURGERY

VOL. XVIII.

JULY, 1930.

No. 69.

SOME BYGONE OPERATIONS IN SURGERY.

By SIR D'ARCY POWER, K.B.E., LONDON.

I. CUTTING FOR THE STONE.

CUTTING for the stone by lateral lithotomy may be said to have died with the advent of Listerian surgery. Its career was long and chequered, but the end was glorious, a surgical feat amounting almost to legerdemain. At every hospital a lithotomy was placed first on the operation list. At St. Bartholomew's the operating theatre was crowded with eager students, and the front bench was filled by the whole surgical staff when it was announced that lateral lithotomy would be done. Accurate timing showed that Sir Thomas Smith, Surgeon and Lithotomist to the Hospital, could extract a stone in fifty-nine seconds from the time he took up the lithotomy knife. Sir William Savory took sixty-one seconds, but he was ambidextrous. It was always a matter of speculation whether he would use his left or his right hand, and I remember at least one patient who had a scar on each side of his perineum. Both surgeons held the knife in their teeth, the last remnant perhaps of an age-long custom—a vestigium of the time when the fingers of the left hand were engaged in the rectum whilst those of the right were employed in pressing down the stone through the abdominal wall. For more than a hundred and fifty years lateral lithotomy had been the great show test of a skilful surgeon. Cheselden had perfected the operation, and only a few of his successors had approached, and none had beaten, his record of forty-five seconds from start to finish.

There were abundant opportunities for display. Stone in the bladder was very common, more so in some districts than in others. There were no cottage hospitals, and the sufferers were therefore obliged to go to the large towns for operations. Norwich and London were especially favoured, and at the Norfolk and Norwich Hospital it was not unusual for a surgeon to cut several patients for stone in a single day, and with surprisingly good results.

CUTTING ON THE GRIPE, THE LESSER OPERATION, THE APPARATUS MINOR.

The earliest operation of cutting for stone was the simplest. It was known in later times as the 'apparatus minor' because it only needed a knife

and a hook. But from time immemorial it was called in England 'cutting on the gripe' because the stone was made to project into the perineum by pressure of the fingers of the operator in the rectum. It remained in favour



FIG. 1.—Cutting on the gripe by the apparatus minor.

in Europe until the middle of the sixteenth century, and is probably still performed by native lithotomists in the more remote parts of India. The operation was performed from choice upon children. The perineum was well rubbed with oil or ghee until it became soft and flexible. The stone could

then be felt distinctly, and by the pressure of two or at most of three fingers (*Fig. 1*) passed through the anus into the rectum could be made to protrude at the point most convenient for incision. The knife was made to cut transversely across the middle line in the original operation, or, in later times, on the left side "the breadth of a grain of wheat" away from the raphé. No staff was used as a guide, and if the stone did not at once protrude it was drawn out by a hook with a roughened end.

Monsieur Tolet, Lithotomist in the Hospital of the Charity at Paris, gives the following account of an operation performed by this method in 1680. He says: "There was brought to Mons. Jonnot at La Charité a boy of seven years of age who had been cut the year before. He needed not to make use of the catheter to assure him of the distemper for with two fingers one might feel the stone towards the middle of the perinæum above the former cicatrix. To hold it fast it was needless to put the fingers in the anus because the stone being too high it could not be compressed, as is usually done, when it is in the neck of the bladder. But having caused the scrotum to be lifted up and the skin held straight, Mons. Jonnot turning the inside of the left hand towards the cods, pressed the tumour with his thumb and forefinger on both sides of the suture [raphé] where he had observed the stone. At the same time he made the incision on the side of the suture upon the tumour and, having opened a passage, with a middle-sized hook he extracted the stone; afterwards feeling with one finger that there were others still remaining he was obliged to keep the place dilated with the same finger which introduced the hook and, that the stone might not shift its place, he held it fast with the forefinger of the left hand put upon the upper part of the wound and with the thumb on the lower part. By this means he happily finished the operation that he might afterwards cut the same patient by the great operation; for having pulled out all the small stones and fragments remaining in the perinæum he introduced by the yard a furrowed catheter wherewith he found another stone in the Bladder."

THE MARIAN OR GREATER OPERATION, THE APPARATUS MAJOR.

The Marian operation was so called because it was popularized by Marianus Sanctus Barolitanus in 1535, though it was invented by Joannes de Romanis, a surgeon of Cremona, in 1520. It was truly named lithotomy by the apparatus major on account of the number of instruments required and the severity of the whole procedure. It was, however, extensively practised for many years and was the forerunner of the more modern lateral lithotomy. It was essentially a tearing or rending operation and was looked upon with dread both by the surgeon and the patient.

Preliminary Preparation.—"Before the operation", says Tolet, "the patient ought to be prepared for some days by a Diet, Bloodletting, Glysters and Purges reiterated more than once according to the advice of a skilled physician. And the night before the operation the patient must take a laxative or else an astringent glyster according as need shall require. He must have a day's time at least to rest in after he hath taken physic, and spiritual

THE BRITISH JOURNAL OF SURGERY

remedies are not to be omitted. At the time of cutting the Operator must choose four faithful servants at least, a Chamber indifferently warm and in it good air where the light of day may suffice. He must have an apron and sleeves and under the table a tub of warm water to wash the instruments; upon the table there must be salad-oyl or oyl of Roses and there he is to lay his instruments in order for the Operation. The operation is somewhat terrible to the patient and there are some operators that hide their instruments in their pockets or put the catheters in their button-holes before they anoint them with oyle; others lay them in a dish, nevertheless seeing it makes a noise when the operator takes them up to operate with, it seems more commodious to have them in a pouch or Budget. It is fitting, too, that the Chirurgeon should speak to the patient but in few words, that he look upon him and encourage him with discourse, shunning everything that is undeeent because one must be very reserved to oblige those that are present to be silent and with respect to be attentive, which wonderfully comforts a patient;

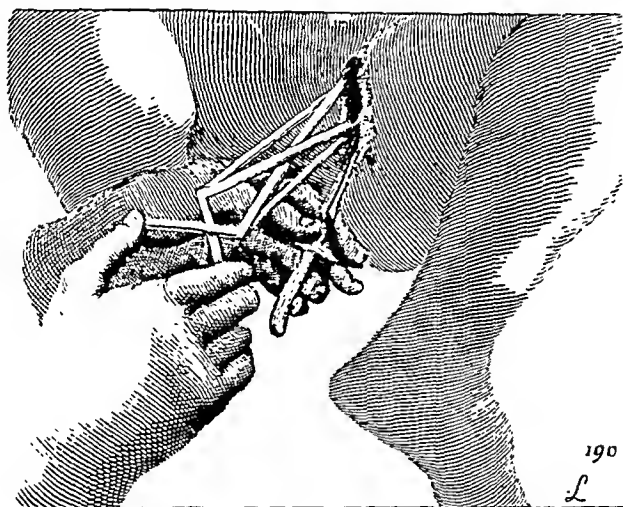


FIG. 2.—The Marian operation by the apparatus major.

calling to mind that to laugh at the disease, the infirmity or expressions of a patient is to insult over a man in affliction. *In calamitoso risu etiam injuria est.* It were better to have but few spectators because a great many may incommode the Operator, frighten the patient and put a wrong construction upon the accidents that sometimes accompany the operation—either designedly or because they are bad judges—. It is fitting, as much as conveniently may be, that a Churchman should be present to talk now and then to the patient as it may help to divert his pain and increase his patience during the operation."

The Operation.—The operation was performed by introducing a grooved staff into the bladder and cutting down upon it to one side of the middle line without dividing the transverse perineal muscle. The ischiorectal fossa, therefore, was not opened, and the space through which the stone could be

removed was unnecessarily limited. The groove in the staff was then felt for and the membranous urethra opened by fixing the point of the knife in the staff. A probe was passed along the knife into the groove and so into the bladder. The staff was withdrawn and two strong iron probes called 'the conductors' were handed to the surgeon. The female conductor was grooved, the male had a probe point. The female conductor was passed along the probe already in the bladder and the male conductor was slid along its groove until it too was in the bladder, the probe first passed being then withdrawn. The surgeon took a conductor in each hand and by forcible separation tore through the deep parts, including the prostate. He next passed a dilator into the bladder between the two conductors and stretched the wound still more until the forceps could be introduced to seize the stone. If the stone were found to be too large for extraction, two 'supporters or latera' were passed by the side of the forceps and were locked upon the stone to form a four-bladed instrument. (*Fig. 2.*)

The operation was rough, cruel, and prolonged, yet it remained in use for very many years and was apparently fairly successful. It is said that "there are some patients who by reason of a good constitution of Body are cured in eight, fifteen or twenty days but the ordinary time of curing Lithotomy is thirty or forty days."

After-treatment.—This consisted "in abstaining from wine until the seventh or eighth day unless the patient be very weak. He is to use a Tisane made of the shavings of Hart's Horn and Ivory or with a little Linseed and the roots of mallows and marsh-mallows or, at least in time of need, he is to drink chalybeate water or of the Tincture of Red Roses, yet not according to his thirst but a little less for fear of a looseness. It is enough that in four and twenty hours he take five or six messes of Broth and a few eggs. Blood-letting and cooling and anodyne glysters are good if he have a fever and pain in the lower belly but all by the advice of a Physieian. And after the first seven or eight days are over and the symptoms have ceased his food is to be made stronger beginning with porridge or Panadoes [bread pudding], then a little meat and bread after that he hath been purged which is commonly to be done about the fourteenth or fifteenth day after the operation."

François Tolet, from whose book the illustrations are taken, was a pupil of Jonnot, Surgeon to La Charité in Paris, and became Lithotomist to the King. His *Traité de la Lithotomie* was translated into English by A. Lovell in 1683. The English translation is dedicated to "The Ingenious and expert Chirurgeon Mr. Tho. Hobbs Lithotomist in the Hospital of St. Bartholomew in London".

ENDOTHELIOMA OF THE CAUDA EQUINA.**REPORT OF A CASE WITH REMOVAL OF TUMOUR AND RECOVERY.**

BY CECIL P. G. WAKELEY, C. WORSTER-DROUGHT, I. M. ALLEN,
AND W. E. CARNEGIE DICKSON,

FROM THE WEST END HOSPITAL FOR DISEASES OF THE NERVOUS SYSTEM, LONDON.

THE case here described presented several interesting features which render it worthy of record. The symptoms of the cauda equina tumour were unusual, the diagnosis of the condition and the localization of the lesion presented certain difficulties which were overcome only by complete investigation, and the result of the removal of the tumour was particularly satisfactory. Finally, the exact nature of the tumour was for some time a matter of conjecture; in fact, its classification is still open to discussion, as it showed features suggestive of a fibroma, of an endothelioma, and of an angioma. The final decision rested between the last two, but the balance of evidence appears to support a pathological diagnosis of endothelioma.

CASE REPORT.

CLINICAL FEATURES.—I. T., male, age 54 years, was admitted to hospital on April 6, 1929, complaining of pain in the left side and left thigh, with weakness of the left leg of nine months' duration.

The onset of the symptoms was gradual. When the patient began to walk, pain appeared in the left gluteal region and in the back of the left thigh. It also appeared when he coughed, and was then usually associated with a spasmodic movement of the left leg. It was worse in cold and damp weather. Later, a dull ache was present continually, and during acute exacerbations the pain behind the left hip travelled down the back of the left thigh, and then down the front and back of the left leg as far as the ankle. The constant ache and the acute exacerbations became gradually worse until about one month before admission, when they were less severe.

Sleep was variable. The patient was extremely irritable at the onset of the illness, but had become less so recently. There was some delay in beginning micturition; the bowels were free with aperients. All other functions were normal. The patient had lost much weight, but recently had gained some. The previous history was uneventful. The patient's first wife died of diabetes mellitus at the age of 40. By his second marriage there was one child, age 6 years, said to be somewhat backward.

ON EXAMINATION.—The right pupil appeared to be slightly smaller than the left, and reacted sluggishly to light and consensual stimulation. It reacted briskly to accommodation, and the left pupil reacted briskly throughout. The vision, visual fields, optic discs, and ocular movements were normal.

There was no nystagmus. Speech and hearing were normal. Nothing abnormal was found in the other cranial nerves.

Sensation.—Spontaneous abnormal sensations included a dull ache in the left gluteal region and left thigh, together with occasional acute exacerbations of pain from the back of the left hip, down the back of the left thigh, and into the front and back of the left leg. In the upper limbs and trunk all types of sensation were normal. In the lower limbs cotton-wool, pain, and thermal sensations were normal. The left tendo Achillis and the muscles of the left calf were more sensitive to deep pressure than were those of the right leg. Vibration sense was diminished in both the right and left tibiae. Postural sense was normal.

Upper Limbs.—Motor power, muscle tone, and position were normal; there was no wasting; and the tendon reflexes were brisk and equal on the two sides.

Lower Limbs.—The left leg and thigh were wasted in comparison with the right, the left leg being one-eighth of an inch and the left thigh one-quarter of an inch less in circumference than the right. The left gluteal region was definitely wasted, with obliteration of the gluteal fold. Motor power in the right leg was normal, but in the left it was weak for all movements. The muscles of the left leg, and especially those of the left gluteal region, were flabby compared with those of the right leg. The right knee-jerk was more active than the left; both ankle-jerks were absent; there was no ankle clonus; both plantar reflexes resulted in plantar-flexion.

The abdominal reflexes were present, but sluggish. The patient walked with a limp, the left hip- and knee-joints being slightly flexed.

Spontaneous Movements.—The muscles of the left gluteal region, and both right and left calf muscles, showed rapid wavy movements of fibrillation. Direct percussion of these muscles resulted in a slow wavy contraction, in contrast with the brisk contraction elicited elsewhere.

Co-ordination was normal. Beyond slight delay in emptying the bladder, the sphincters were also normal. Apart from the presence of one septic tooth-root the digestive system was normal. The bowels were regular with aperients.

Heart.—The apex beat was in the normal position, but the impulse was very faint. The sounds were regular and distant. The pulse was small and regular. The blood-vessels were normal. The blood-pressure was 170 systolic and 110 diastolic.

Vasomotor System.—There was redness of the skin, with a slight papular eruption, over the third, fourth, and fifth sacral areas on both the right and left sides.

Respiratory System.—The chest was large and barrel-shaped; expansion was poor, and the breath-sounds were faint. There was slight tenderness over the lower part of the sacrum.

SUBSEQUENT INVESTIGATIONS.—X-ray examination of the spine was negative.

Cerebrospinal Fluid.—On lumbar puncture in the third lumbar interspace, cerebrospinal fluid was withdrawn in three specimens: (1) 1.5 c.c. of slightly yellow fluid, resembling a pale urine in colour; (2) 6 c.c. of perfectly

clear, colourless fluid; and (3) 4 e.c. of perfectly colourless fluid. The first specimen was contaminated with 73 red blood-cells per e.mm., the second with 5, and the third with 1. In the first specimen there were 18 white cells per e.mm., in the second 4, and in the third 3. The cells in the first two specimens were almost all 'small lymphocytes', with only a very occasional polymorph. The total protein content of the three specimens was: (1) 0.4 per cent; (2) 0.35 per cent; (3) 0.25 per cent. Globulin was much in excess in all three specimens. Fibrinogen and euglobulin were present in moderate quantities in No. 1, in small quantities in No. 2, and absent from No. 3.



FIG. 3.—Skiagram showing lipiodol held up with a horizontal lower border opposite the middle of the body of the 3rd lumbar vertebra.

Pseudo-globulin was present in large amounts in all three specimens, but slightly less in No. 3 than in the other two. The Lange test resulted as follows: in No. 2, 3454432100, and in No. 3, 3444432100. The Wassermann reaction in both the blood and the cerebrospinal fluid was negative.

By cisternal puncture 2 e.c. of blood-stained fluid were withdrawn, and 1.5 e.c. of lipiodol were introduced into the cisterna magna. X-ray examination showed that the lipiodol was held up with a horizontal lower border opposite the middle of the body of the 3rd lumbar vertebra (*Fig. 3*).

OPERATION (May 1, 1929).—A zig-zag incision was made with its transverse part opposite the spine of the 3rd lumbar vertebra. The vertebral column was exposed in the usual way.

The spines of the 2nd, 3rd, and 4th lumbar vertebrae were removed. The vertebral arches were removed at this level, and at a later stage the wound was enlarged one space further down in order to secure better exposure. On palpation of the dura a slight firmness was felt at and below the level indicated by the lipiodol. The dura was opened by a longitudinal incision. On separating the fibres of the cauda equina, a slightly pear-shaped, semi-cystic tumour was found lying longitudinally in front of the cauda equina rather more towards the left side than the right, the upper pole being opposite the middle of the body of the 3rd lumbar vertebra (*Fig. 4*). The tumour was separated easily from the surrounding structures, but

remained attached by a firm strand at its upper pole and by another at the middle of the left lateral border. These strands were divided with scissors. The dura was not closed. The superficial structures were sutured, the cavity being drained by a rubber drain.

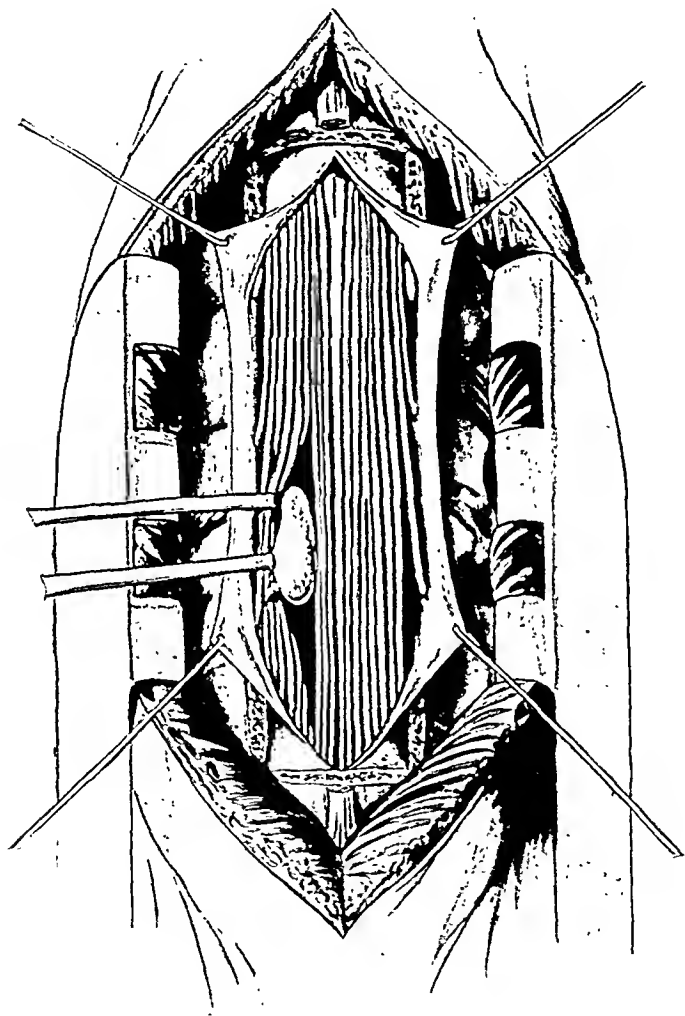


FIG. 4.—Representation of tumour *in situ* at the time of operation. The pear-shaped and semicystic tumour is lying longitudinally in front of the cauda equina, rather towards the left side, the upper pole being opposite the middle of the body of the 3rd lumbar vertebra.

SUBSEQUENT PROGRESS.—There was a free discharge of cerebrospinal fluid from the wound for two days after the operation. Retention of the urine occurred for the first two days, necessitating catheterization; but control of the bladder gradually increased until the seventh day, when the patient

was able voluntarily to pass twenty-two ounces of urine. On the fifteenth day a small collection of blood-stained serous fluid was evacuated from beneath the middle of the wound. The patient was then passing urine freely.

On the eighteenth day all the movements of both lower limbs were performed satisfactorily, and with massage and exercises the wasted muscles were regaining their tone. All types of sensation in the lumbar and sacral segmental areas were normal. The right knee-jerk was still slightly more active than the left, but the difference was much less than before. The right ankle-jerk was then present, but the left was still absent. Both plantar reflexes were flexor. The bladder was acting normally.

On the twenty-third day the patient was well except for slight constipation. There was some irritation at the end of the penis during micturition, probably due to the hexamine which had been given as a prophylactic against bladder infection. On the twenty-eighth day the patient was up for four hours and was able to walk around the ward comfortably.

Five weeks after the operation there was slight aching in the small of the back. There had been no further pain in the legs. The patient was able to walk freely. The muscles of the left gluteal region and of both calves were quite firm and strong. There was still a very slight difference in the knee-jerks, the right ankle-jerk was more active, but the left was still absent. Later the left ankle-jerk returned, but was still very sluggish.

PATHOLOGICAL EXAMINATION OF THE TUMOUR.—The mass removed was roughly about the size and shape of an almond, weighted 2·3 gm., and measured 28 mm. long by 15 mm. broad by 10 mm. in depth. It had a

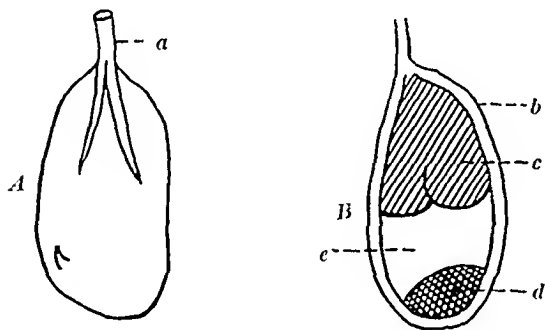


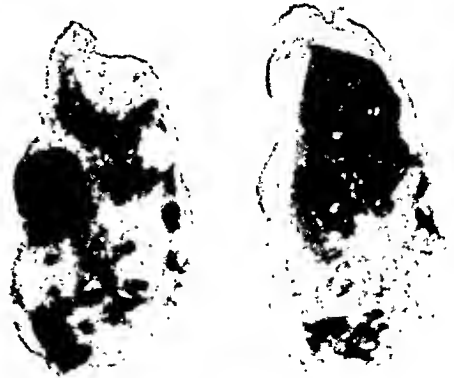
FIG. 5.—A, The tumour: a, Thin bifurcating fibrous cord attached towards upper end of tumour. B, Appearance of tumour on vertical section. b, Thick fibrous capsule; c, Recent blood-clot in upper half; d, Smaller blood-clot at lower pole; e, Pale yellowish central tissue, translucent in parts, suggesting mucoid to slightly fatty tissue with small cystic spaces.

thick fibrous-tissue wall, attached to which towards the upper end was a thin bifurcating cord which appeared to be fibrous tissue. A similar small tag was attached towards the left postero-lateral margin. These cords resembled a nerve or a small thickened vessel, and subsequent microscopical examination proved them to be the latter.

On vertical section the appearances were as in the diagram (*Fig. 5*), a thick, fibrous capsule with a larger mass of blood-clot in the upper half, and a smaller similar mass at the lower pole. The central tissue between had a somewhat variegated appearance, mostly pale and yellowish, translucent in parts, suggesting a mucoid to slightly fatty tissue, with small cystic or angiomatous spaces, some with pale contents and others containing blood or blood-clot.

The appearance in further parallel sections is shown in the naked-eye photographs (*Figs. 6, 7*), the former being towards the posterior part of the tumour and showing varying admixtures of the blood-clot-filled spaces and tumour-like tissue.

Microscopical Examination.—The appearances varied very much in different parts of the tumour. The largest space, occupying nearly the upper half of the mass and filled with recent blood-clot (*Fig. 8*), was situated within the somewhat thick and dense fibrous capsule. The inner surface of this wall showed no endothelial lining, but only a rather indefinite layer of flattened connective-tissue cells. The intermediate tumour-like area at some parts suggested a fibroma undergoing patchy and widespread degeneration. Some of the little cyst-like cavities appeared to be pseudo-cysts formed by mucoid



FIGS. 6, 7.—Naked-eye photographs of tumour in parallel sections, *Fig. 6* being towards the posterior part of the tumour and showing varying admixtures of blood-clot-filled spaces and tumour-like tissue. ($\times 2$.)

softening, into some of which hæmorrhages had taken place; others of the spaces were more definite in their outline. At first sight these spaces, especially where they were numerous or close together, were very suggestive of a cavernous hæmangioma or lymphangioma, particularly the latter, as they contained more or less homogeneous lymph-like contents except where hæmorrhages had occurred into them. The area shown in one microphotograph was occupied by an extensive hæmorrhage, surrounded by phagocytic cells containing blood pigment, showing dark in the photograph (*Fig. 9*). On the whole these spaces appeared to be due to pseudo-cystic mucoid softening rather than to the new formation of true angiomatous spaces.



FIG. 8.—Microscopic section of tumour (van Gieson's stain).

In some parts of the tumour there were endothelioma-like areas situated round the numerous thin-walled blood-vessels, i.e., a peritheliomatous formation (*Fig. 10*).

The whole structure was, therefore, extremely variegated and somewhat difficult to interpret. It probably originated as an endothelioma in the walls of one of the small vessels on the surface of the cauda equina. As this enlarged, it became surrounded by a fibrous capsule. Some parts had undergone mucoid or 'myxomatous' degeneration and softening with the formation of pseudo-cysts, so numerous in places as to simulate a cavernous angioma with numerous patchy hæmorrhages, large and small.

COMMENTARY.

The case was that of a man of 53 who had suffered from pain in the left gluteal region, the back of the left thigh, and the front and back of the leg as far as the ankle, constantly and in attacks, for a period of nine months. The pain seemed to involve the whole of the sacral segmental areas and the 5th lumbar area as well, a fact which in itself suggested that it was symptomatic of a lesion of the posterior nerve-roots or of the cauda equina. Apparently when the patient first came under medical observation a provisional diagnosis was made of sciatica on the character of the pain and the absence of signs elsewhere indicating involvement of the nervous system at a higher level.

When the patient came under personal observation the symptoms had not changed except for a slight amelioration of the pain. The physical signs had, however, varied

FIG. 9.—Section of tumour where an extensive hæmorrhage was surrounded by phagocytic cells containing blood-pigment showing dark to the microscope.

considerably. In the left leg, in addition to the pain there was pronounced wasting of the left gluteal muscles and slight wasting of the calf muscles, in association with sluggishness of the knee-jerk and absence of the ankle-jerk—all signs which might be explained by involvement of the lumbar and sacral nerve-roots. In addition there were fibrillary contractions in the two groups of wasted muscles. Examination of the right leg, however, demonstrated that the ankle-jerk on that side was also absent, that the muscles of the calf were slightly flabby, and showed occasional fibrillary contractions.

The absence of both ankle-jerks at once suggested that the lesion was placed at a higher level than was at first suspected, while the appearance of fibrillary contractions in the muscles of both calves indicated that the lesion must be one

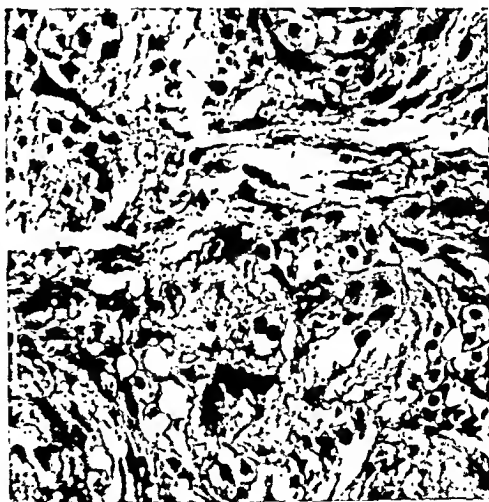


FIG. 10.—Section of tumour showing endothelioma-like areas situated round thin-walled blood-vessels of peritheliomatous formation.

interfering with motor fibres. The sacral form of *tabes sacralis* appeared to be excluded by this fact and by the character of the pain. Osteo-arthritis of the lumbosacral spine was excluded by the negative X-ray examination of the spine and possibly by the fact that after nine months the pain still remained confined to the left side.

It then appeared to be reasonable to place the lesion responsible for the symptoms in the cauda equina. The absence of symptoms of bladder involvement of any degree suggested that the lesion was low enough to allow the *conus medullaris* to escape. On the left side the sensory roots involved were the 5th lumbar and at least the upper sacral, while the motor roots were chiefly the 3rd and 4th lumbar and the 1st sacral. On the right side the 1st sacral appeared to be the only motor root involved, while there was no interference whatever with the sensory roots. Hence, on the clinical investigation, there appeared to be a tumour of the cauda equina within the vertebral canal, to the left side rather than to the right, and, since on both sides the motor functions were more involved than the sensory, the lesion probably lay in front of rather than behind the roots of the cauda equina. A remarkable feature of the case was that the pain was confined to the left side throughout.

Since the lesion occupied the whole or the greater part of the spinal canal from side to side, it was probable that some evidence of spinal block would be found. Lumbar puncture was performed below the level at which the tumour was suspected to lie, and changes in the cerebrospinal fluid indicative of a block were found on detailed examination. Certain interesting points in connection with the cerebrospinal fluid changes were as follows: (1) The first specimen of cerebrospinal fluid withdrawn was slightly yellow, but the others were colourless. (2) The percentage of the total protein steadily decreased from 0.4 per cent in the first specimen to 0.25 per cent in the last specimen. (3) The fibrinogen and euglobulin and pseudo-globulin steadily decreased in amount from the first specimen to the last. (4) The yellow colour of the fluid was associated with a protein content of 0.4 per cent, moderate amounts of fibrinogen and euglobulin, and large amounts of pseudo-globulin; but with 0.35 per cent of protein, small quantities of euglobulin and fibrinogen, and large quantities of pseudo-globulin, no yellow coloration occurred. Reference to our records shows that in no case has a yellow colour of the cerebrospinal fluid been present with a total protein content of below 0.35 per cent. (5) The Lange colloidal gold test showed a maximum decoloration in the tubes to the left of the middle of the system—a pseudo-tuetic curve. (6) The findings in the cerebrospinal fluid in this case and in others in which spinal block was present have been duplicated in every particular by cases in which extensive cerebral and spinal thromboses have been present, and simulated in a lesser degree by cases in which the vascular lesion was less extensive. It would appear that the pathological basis of the findings was the same in both groups of cases—the lesion leading to an increased flow of proteins and globulins from the blood-stream to the cerebrospinal fluid, in the thrombotic cases as a result of the thrombosed area bordering on the cerebrospinal fluid system, and in the cases of spinal block as a result of venous stasis below the level of the block.

These observations on the cerebrospinal fluid would appear to have certain practical applications: (1) When the clinical history or the physical signs suggest in any way that a spinal block may be present, it is advisable to collect the cerebrospinal fluid obtained on lumbar puncture as a number of small specimens, not only to eliminate the possibility of contamination of the whole specimen with red blood-cells, but also to reveal the special findings indicating spinal block at their maximum. (2) The colour of the fluid cannot be relied upon as a sign of spinal loculation, as it appears only when the total protein and globulin have increased to large quantities. It must not be expected that the fluid should be yellow when the protein and globulin are small in quantity. Probably the French name for the changes which are present in cases of spinal block, proteo-cytological dissociation, *dissociation albumino-cytologique*, would indicate the changes most often found rather than the usual description of Froin's syndrome. (3) In patients in whom there is evidence of vascular degeneration and there are no definite indications of block, the appearance of all the cerebrospinal fluid findings usually associated with a block cannot be accepted as unequivocal evidence of an obstruction.

In order that the position of the tumour might be more accurately determined, a cisternal puncture was performed, lipiodol introduced, and a skiagram of the lumbosacral spine taken. This showed that the lipiodol was completely held up with a definite horizontal lower border opposite the middle of the body of the 3rd lumbar vertebra. This observation was accepted as evidence of the presence of an obstruction at and below that level, although it was not possible to correlate it with the impression gained from the examination of a number of skiagrams after the intrathecal introduction of lipiodol in cases in which the obstruction was at a higher level—namely, that with intramedullary lesions the lower border of the lipiodol shadow was horizontal, whereas with extramedullary lesions it was more often in two parts, one at a higher level than the other. Incidentally, in cases of tumour of the cauda equina, the use of lipiodol may be of real value in localization, and essential to confirm the indications for operation. Quite frequently, from the clinical features alone, all that can be concluded is that the lesion does not involve roots above a certain level; whereas in cases in which some obstruction exists the use of lipiodol decides the exact level. There are not present the same sources of error in interpretation as when the obstruction involves the spinal cord, lipiodol having been observed to be held up not only by intramedullary and extramedullary tumours, but in cases of transverse myelitis, disseminated sclerosis, spinal thrombosis, and thickening of the dura.

The successful outcome of the operation depended upon a series of factors: (1) The complete localization of the tumour before the operation was undertaken. (2) The consequent reduction in the handling of the roots of the cauda equina to a minimum and the lessening of the risk of injury to the nervous tissue. (3) The fact that the fibres of the cauda equina were interfered with by pressure alone and were in no way involved in the tumour. Interference with the functions of the bladder, a common cause of failure in operations on the cauda equina, occurred for only a short period immediately after the operation, and then disappeared entirely. A feeling of weakness in

the back was still a source of discomfort to the patient at the time of discharge from hospital, but this symptom disappeared within a few weeks.

The nature of the tumour removed has been discussed in detail on pp. 10, 11. As stated there, the appearances suggested in parts a fibroma, in others an endothelioma, and in others an angioma. The evidence, however, appeared to support the diagnosis of an endothelioma to the exclusion of angioma.

SUMMARY AND CONCLUSIONS.

1. A case of endothelioma of the cauda equina is described in detail, and all aspects of the clinical and pathological picture are discussed.

2. The symptoms and signs of the cauda equina lesion *per se* were distinctly unusual.

3. The appearance of bilateral signs of any kind, whether sensory, motor, or variations in the reflexes, in the course of a case which appears to be one of sciatica, should at once suggest that a lesion is in the wall or lumen of the spinal canal, and further investigations should be made to decide the nature of the lesion.

4. The changes in the cerebrospinal fluid suggesting spinal block are discussed, and attention is drawn to the fact that in early cases, or those in which the abnormalities are slight, the changes may be more obvious in the earlier specimens of fluid withdrawn than in later specimens.

5. Attention is drawn to the fact that the cerebrospinal fluid changes considered characteristic of spinal block may be simulated in cases in which there are thrombotic lesions of the brain or spinal cord possibly in close contact with the cerebrospinal fluid system.

6. The yellow colour of the fluid in cases of spinal block and in those of thromboses would appear to be found only when protein and globulin are present in large quantities.

7. The use of lipiodol and skiagraphy is of definite value in the investigation and localization of lesions of the cauda equina, and is probably more useful and less subject to errors of interpretation than in cases of spinal block involving the spinal cord.

8. The surgery of lesions of the cauda equina is facilitated by a detailed investigation of the patient and localization of the lesion by all available means before the operation is undertaken.

9. The nature of the tumour removed is discussed in detail and the conclusion reached that it is probably an endothelioma growing from a small vessel in the cauda equina, rather than an angioma.

HERNIA IN AFRICANS.

By W. K. CONNELL,

EAST AFRICAN MEDICAL SERVICE, DAR-ES-SALAAM.

THE tendency to blame modern civilization for most of mankind's ills has gone much too far. We are assured by various authorities that cancer is mainly the result of an artificial diet and that it could not occur amongst plain people eating plain food in a plain way. This view is not borne out by the facts. There is no dearth of malignant disease amongst even the most primitive of our East African tribes. In the same way we have been asked to believe (despite the overwhelming evidence in support of Hamilton Russell's 'saccular theory') that a potent cause of rupture is the use of a water-closet with an excessively high seat. There is little support for such a theory. I cannot quote statistics, but I feel sure that my colleagues in East Africa will support me when I aver that rupture is much commoner amongst East African natives than amongst the inhabitants of the British Isles; and this notwithstanding the fact that the East African answers all Nature's calls in the prophylactically ideal squatting posture.

Personally, I believe in Hamilton Russell. I think that if you have no congenital sac, the danger of hernia is negligible; and, conversely, that if you *have* a congenital sac, the bowel or omentum will enter it despite your utmost precautions.

Between Sept. 5, 1927, and Nov. 5, 1929, I have personally operated on 208 cases of hernia at the Sewa Hadji Hospital, Dar-es-Salaam, and a brief summary of these cases may be of interest to my fellow-practitioners and may reveal a few facts of importance:—

Right Inguinal Hernia, 142: Of these, 18 were of the 'sliding' type, 9 were strangulated, 5 recurred, 3 died (from shock, sepsis, and faecal fistula respectively), and in 7 the operation was abandoned (6 because the hernia was of the extreme 'sliding' type, and 1 because it was irreducible owing to adhesions).

Left Inguinal Hernia, 63: Of these, 2 were strangulated, 2 recurred, 2 died (1 from secondary strangulation, and 1 after resection of the gut), and in 3 the operation was abandoned (owing to irreducibility from adhesions, collapse under the anaesthetic, and lymphorrhœa).

Umbilical Hernia, 1: This case was strangulated.

Ventral Hernia, 1.

Right Femoral Hernia, 1.

The summary speaks for itself, but a few additional or explanatory remarks are called for:—

Of the inguinal hernias 18 were bilateral. As until recently I had no idea of publishing my cases I unfortunately omitted to mention in the operation register which of the hernias were 'direct' and which 'oblique'.

The vast majority were certainly 'oblique'. Right inguinal was more than twice as common as left inguinal hernia.

Of the right inguinal hernias 14·08 per cent were of the 'sliding' type—a variety seldom found in Britain, and scarcely mentioned in the text-books. These cases are very troublesome; and I have been utterly defeated in 6 out of the 18 which I have treated, and have had to abandon the operation. What is one to do when the whole of the ascending colon is in the scrotum and in consequence the posterior aspect of the hernial mass is for the most part devoid of a peritoneal covering? Would one be justified in performing an excision of the proximal colon and implanting the ileum into the transverse colon? I doubt it. Some of these cases are a veritable trap, particularly where only the attenuated tip of the cæcum has been dragged down on the posterior wall of the sac. It is easy to overlook the true nature of these cases and to include the lower part of the cæcum in a ligature round the sac neck—with disastrous results to the patient.

Why is 'sliding' hernia so unusually common in East Africans? Can it be due in some measure to gaseous distension of the cæcum resulting from an almost purely vegetarian diet?

Strangulation had occurred in 6·33 per cent of the right and 3·17 per cent of the left inguinal hernias. The only umbilical hernia in the series was strangulated. Only one death occurred in the total series of 12 strangulated hernias, and this death followed an extensive resection of the gangrenous bowel with drainage of the divided ends. Ten operations were abandoned for various reasons.

The total mortality for the whole series of hernias (including the strangulated cases) was 2·4 per cent. The causes of death were: Collapse following resection (1); ordinary post-operative shock (1); secondary strangulation due to a small loop of bowel bursting through the purse-string sutures used to close the neck of a gigantic left-sided inguinal hernia which had contained practically the whole of the patient's intestines (1); exhaustion from fecal fistula (1); sepsis (1).

Perhaps the most striking fact brought out by this series is the apparent immunity of the East African female to rupture. Thus, of the total of 208 cases, only 2 occurred in women—one being an umbilical and the other a femoral hernia. Last tour my experience was similar, as I saw but one case of hernia in a native woman.

It will be interesting to learn if my experience of the seeming rarity of rupture in East African women is shared by my colleagues in this territory. Is rupture in these women really uncommon, or does feminine modesty prevent the patients from displaying their disability to a European M.O.? Such diffidence would hardly survive the pangs of strangulation, and this inclines me to believe that native women really *are* astonishingly immune to rupture—especially in view of the fact that strangulation is commoner in femoral than in inguinal hernia. What is the explanation of this immunity? The magnificent physique of most East African women may possibly be one factor, for on the whole the native woman is better built and has a better carriage than the native man. Furthermore, in her work she is not usually subjected to the sudden strains which affect the male labourer, for the European

conception of the African woman as a universal beast of burden is largely erroneous. If, however, we accept Hamilton Russell's views, we must conclude that, for some reason, the African female is seldom born with a congenital sac.

A very large proportion of the hernias in this series were complicated by other pathological conditions—serotal elephantiasis, hydrocele, hæmatocele, varicocele, filarial cystic enlargement of the cord lymphatics, urethral stricture, and perineal urinary fistula. I have found it best, as a rule, to deal with the hernia alone at the first operation and to operate on any complicating condition at a later date. The danger of sepsis (the chief factor in causing relapse in hernias treated by operation) is thus much diminished.

Some degree of mild septic infection has unfortunately occurred in a fair proportion of my cases. In spite of one's utmost care, first-intention healing cannot always be obtained, and this is particularly true of operations on patients who have suffered severely from ankylostomiasis or whose hernias are complicated by serotal elephantiasis. It is, of course, well known that streptococci often lurk in the tissues of patients with elephantiasis, and I think that the trauma of operation stirs these streptococci into activity.

How to get old hookworm patients into a fit state for operation is a big problem and one which I have not yet solved. Their anemia persists, without apparent improvement, long after the original infestation of the gut has been eradicated. Step-ladder blood transfusions might be of service, and I hope to give them a trial shortly.

One very common source of sepsis is the serotal hæmatoma which inevitably follows the removal of a huge hernial sac. Drainage, even with large rubber tubes, does not prevent this complication, and the introduction of gauze packing makes subsequent infection certain—even though the packing is removed at the end of twenty-four hours. I used frequently to leave the sac behind undisturbed in the scrotum, after having divided and ligatured the sac neck; but the results of this procedure have been uniformly bad, and I have abandoned it. The sac, when left behind, becomes transformed into either a hæmatocele or a hydrocele, in the ultimate treatment of which a hæmatoma is just as certain to form as it would be in a complete one-stage operation for the hernia; so that the complication is only postponed, not avoided.

There is nothing original in the method by which I have operated on these hernias. After splitting the external oblique, I twist the sac neck three or four times, transfix and ligature it as high as possible, and then cut through the sac half an inch below the ligature. I suture the conjoined tendon to Poupart's ligament *in front* of the cord, and close the external oblique in the usual way. Owing to the enormous thickness of the cord in many of these African hernias, it is often impossible to effect anything like a complete closure of the inguinal canal and external abdominal ring. One could, of course, excise most of the constituents of the cord, thus lessening its dimensions; but such a procedure would involve the division of many lymphatic vessels and might determine the development of serotal elephantiasis in a patient already infected with filariæ. In patients with enormously thick cords, Bassini's operation would be impossible. In any case, as Gallie, of

Toronto, has pointed out, it matters little whether the conjoined tendon is stitched to Poupart's ligament in front of or behind the cord, because in either case the union is of an entirely temporary nature. It is this fact, of course, which has led Gallie to employ living sutures (fascia lata strips) in his hernia operations; and I intend to put his method to the test next tour. I believe it is the only method likely to produce good lasting results in the treatment of those enormous hernias with which we in East Africa are so painfully familiar.

Large 'sliding' hernias constitute a baffling problem, the existence of which is conveniently ignored by text-books on operative surgery. Peritoneum is present only on the front, sides, and lower portion of the back of such hernias; it has been my practice to excise the whole of this peritoneum and then to close the 'window' thus formed by means of a purse-string suture inserted round its margins. The result of this manoeuvre is to bend the cæcum and lower part of the proximal colon so that they form a curve with the concavity upwards. If the 'sliding' element is not too extreme, it is then often possible to push the flexed bowel back into the abdominal cavity—though whether or not it is likely to remain there I cannot say. Possibly Gallie's method of closing the inguinal canal might prevent recurrence—especially if the testis and cord were excised at the same time. Where the entire ascending colon has slipped into the scrotum, nothing short of partial colectomy seems likely to be of any use, and I much doubt whether or not such a step would be justifiable. It would appear to the writer that the subject of 'sliding' hernia has not so far received the consideration merited by its prevalence in certain localities and by the peculiar technical difficulties connected with its operative treatment.

The great majority of my patients have had 'oblique' hernias, and their abdominal muscles have usually been very well developed. Both these facts support the view that a congenital sac is the essential cause of East African hernias.

I am not in a position at present to discuss the question of recurrences. None have as yet reported to me, but it is certain that some must have occurred and that more will occur at a later date. In dealing with these enormous African hernias, a considerable percentage of failures must be anticipated.

My excuse for the foregoing paper is its practical bearing on one of the commonest disabilities which East African doctors have to treat.

My thanks are due to Dr. J. O. Shircore, C.M.G., Director of Medical and Sanitary Services, Tanganyika Territory, for permission to submit this article for publication.

CYSTS OF THE LONG BONES OF THE HAND AND FOOT.*

By HARRY PLATT, .

SENIOR SURGEON TO ANCOATS HOSPITAL, MANCHESTER.

INTRODUCTION.

Cysts of the long bones of the hand and foot are recognized as forming a distinct anatomical group in which are illustrated on a miniature scale many of the typical clinical features of bone cysts in general. Thus they tend to develop insidiously, cause little or no discomfort, and often escape recognition until the occurrence of spontaneous fracture, when the diagnosis is immediately suggested by the radiographic appearances. But in the vast literature on cystic affections of the skeleton which has accumulated since the appearance of von Recklinghausen's classical article in 1891,¹ the special characteristics of the cysts which involve the metacarpals, the phalanges, and—more rarely—the metatarsals, have received merely fleeting attention. From time to time isolated cases have been recorded by different writers, but until recently no considerable series has been studied in a comprehensive fashion.

One of the most important of the earlier contributions is an account by Robert Jones and D. Morgan² of four cases, with the histological findings in two cysts which were explored. A number of examples are also quoted by Elmslie³ in the admirable monograph in which he was the first to urge the importance of classifying bone cysts on a definite anatomical and clinical basis. In 1920 Bloodgood,⁴ whose experience of bone tumours and cysts has long been unrivalled, described a group of eight phalangeal cysts which had come under his observation.

On the frequency of the miniature long bone cysts as compared with cysts of the major long bones (humerus, tibia, and femur), we have little statistical information. In 97 bone cysts affecting various sites collected in 1912 by Silver,⁵ there were 7 phalangeal cysts, 1 metacarpal, and 1 metatarsal. In an article published in 1923, containing a very complete bibliography, Roederer⁶ placed the metacarpals and phalanges amongst the rare sites. This is undoubtedly true of the solitary cysts in which the lining and contents show the histological appearances of *osteitis fibrosa*. But when cysts almost identical in clinical and radiographic manifestations, but with a different histogenesis, are included, involvement of the metacarpals and phalanges is, in my experience at least, by no means rare. Thus in a consecutive series of 34 bone cysts operated on during the past nine years, 13 were lesions of the miniature long bones (12 patients). This series does not include operations for true sarcoma, multiple chondromata, or generalized osteitis fibrosa,

* From the Orthopaedic Service, Ancoats Hospital.

in which solitary or multiple cysts of varying size may be incidental. During the same period, 5 additional patients (6 cysts) have come under observation in my clinic in whom no operation was carried out, and therefore a histological diagnosis was not available. It is believed that a consideration of this small but representative group of 17 cases will help to crystallize our knowledge of the pathogenesis, diagnosis, and treatment of the miniature long bone cysts.

Since the greater part of this paper was written, a further important contribution has appeared from the Laboratory of Surgical Pathology of the Johns Hopkins Hospital. Geschiekter and Copeland,⁷ inspired by Bloodgood, have examined the material relating to 175 bone cysts (osteitis fibrosa) and over 200 giant-cell tumours. This unique collection contains a group of 16 cysts of the smaller bones presenting histological features of unusual interest. (*Chondromatous* cysts are deliberately excluded from this survey.)

PERSONAL CASES.

Group I.—HISTOLOGICAL DIAGNOSIS AVAILABLE.

Case 1.—M. McC., female, age 12. Cyst of the proximal phalanx of the right thumb (osteitis fibrosa).

HISTORY.—Swelling of the thumb noticed the day after a slight injury (Aug. 5, 1921).

ON EXAMINATION (Aug. 15).—Thickening and enlargement of the proximal phalanx of the right thumb.

X ray.—Shows multicystic expansion of the proximal phalanx (*Fig. 11*).

TREATMENT (Aug. 19).—*Curettage and cauterization.* The cyst cavity contained soft reddish tissue; no definite lining was noted.

HISTOLOGICAL DIAGNOSIS.—Osteitis fibrosa (*Fig. 12*).

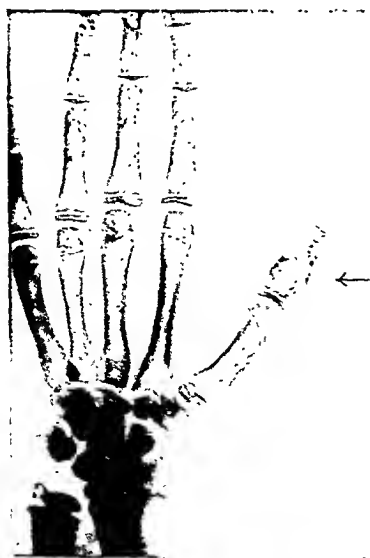


FIG. 11.—*Case 1.* Cyst of the proximal phalanx of the thumb (osteitis fibrosa). Shows multicystic expansion of phalanx. No sign of fracture.



FIG. 12.—*Case 1.* Cyst contents. Bone trabeculae undergoing erosion in the midst of delicate connective tissue. An early phase of osteitis fibrosa.

RESULT.—Six years later—complete obliteration of the cyst by new bone (Fig. 13).

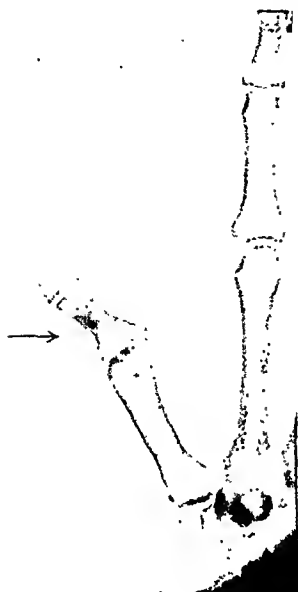


FIG. 13.—Case 1. The affected phalanx six years later. The cystic area replaced by dense bone.

Case 2.—N. D., female, age 15. Cyst of the proximal phalanx of the left little finger—spontaneous fracture (osteitis fibrosa).

HISTORY.—Swelling of the hand followed a direct blow (July 18, 1922). The patient continued work for five days.

ON EXAMINATION (July 25).—Swelling and thickening of the proximal phalanx of the little finger of the left hand; crepitus demonstrable.

X ray.—Shows cystic expansion of the proximal phalanx, non-trabeculated with thin cortex. Incomplete fracture.

TREATMENT (Sept. 3).—Curettage and cauterization. Contents of cyst, semi-translucent 'fibro-cartilaginous' material. Incomplete fibrous lining.

HISTOLOGICAL DIAGNOSIS.—Osteitis fibrosa.

RESULT.—Eighteen months later—cystic area obliterated by new bone.

COMMENT.—The naked-eye appearances of the cyst contents suggested myxochondroma, but the histological picture is predominantly that of osteitis fibrosa.

Case 3.—A. B., male, age 17. Cyst of the proximal phalanx of the left little finger (myxochondroma).

HISTORY.—Fell on the hand whilst playing football (Aug. 18, 1923).

ON EXAMINATION (Aug. 23).—Pain and swelling over the proximal phalanx of the little finger of the left hand. No crepitus.



FIG. 14.—Case 3. Cyst of the proximal phalanx of the little finger (myxochondroma). A trabeculated cyst occupies the distal two-thirds of the phalanx. Shows a fracture through the thin cortex.

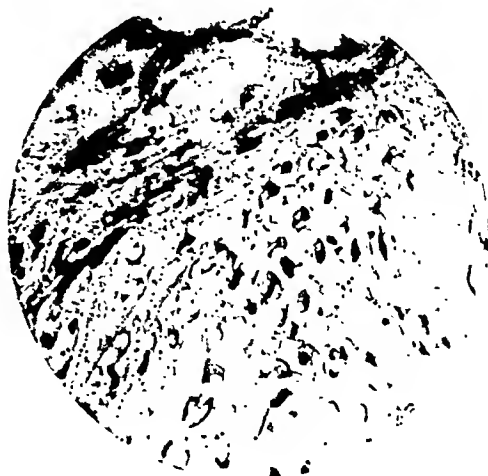


FIG. 15.—Case 3. Cyst contents. Area showing stellate cells.

X ray.—Shows multicystic area in lower two-thirds of the shaft of the proximal phalanx with slight expansion, faint trabeculation, and pathological fracture (Fig. 14).

TREATMENT (Aug. 31).—*Curettage, cauterization, bone-graft.* Cyst interior filled by soft, pale, granular tissue containing glistening nodules. No trace of fibrous lining.

HISTOLOGICAL DIAGNOSIS.—*Myxochondroma* (Fig. 15).

RESULT.—Five years later—phalanx slightly irregular. No disability. Radiograph shows tiny areas of translucency, but the greater part of the cyst has been obliterated (Fig. 16).

Case 4.—H. C., female, age 8. (1) Chondroma of the metacarpal of the right little finger. (2) Cyst of the proximal phalanx of the right little finger (chondroma).

HISTORY.—Swelling developed gradually on the ulnar side of the little finger in the region of the head of the fifth metacarpal.

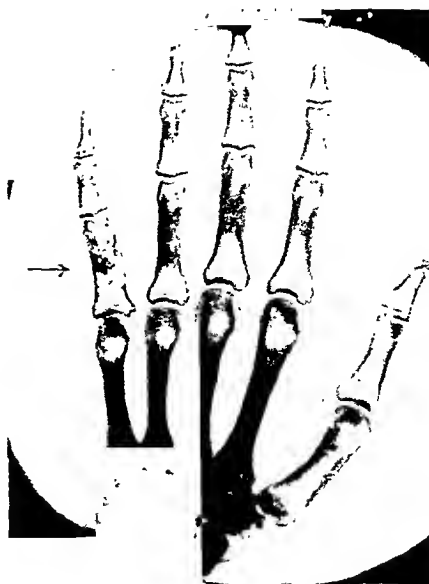


FIG. 16.—Case 3. Five years later. Almost complete obliteration of the cyst; area of translucency still showing.



FIG. 17.—Case 4. Cyst of the proximal phalanx of the right little finger (myxochondroma); solid chondroma of the metacarpal. Shows: (1) Cyst occupying middle third of phalanx, clear-cut outline with thin wall; (2) Tumour arising from interior of neck of metacarpal.

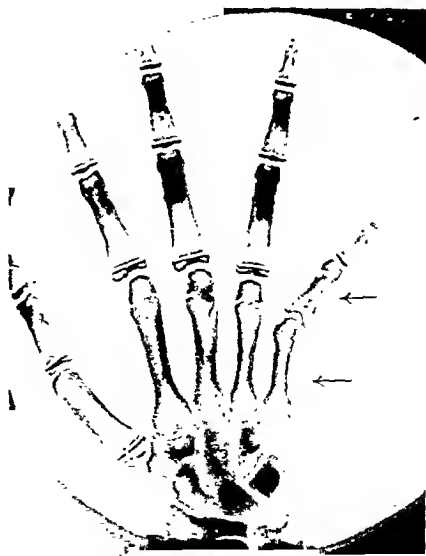


FIG. 18.—Case 4. Five and a half years later. No sign of recurrence in the metacarpal. The phalangeal cyst has been obliterated by dense bone.

ON EXAMINATION (Oct. 3, 1923).—Rounded swelling attached to the head of the fifth metacarpal.

X ray.—Shows: (1) Cystic expansion of the neck of the fifth metacarpal with tumour; (2) Thin-walled cyst in the proximal phalanx, no trabeculation (*Fig. 17*).

TREATMENT (Oct. 8).—(1) *Resection of chondroma from metacarpal.* (2) *Curettage and cauterization of cyst of phalanx, with insertion of bone-graft.*



FIG. 19.—*Case 5.* Cyst of the fifth metacarpal (myxochondroma). Shows cyst occupying the distal half of the metacarpal but not invading the old epiphysis. Fracture has occurred through the base of the cyst.

HISTOLOGICAL DIAGNOSIS.—(1) Metacarpal tumour—chondroma. (2) Cyst in phalanx—myxochondroma, abundant stellate cells.

RESULT.—Five years and four months later—no recurrence of metacarpal tumour; phalangeal cyst obliterated by dense bone (*Fig. 18*).

Case 5.—E. T., male, age 31. Cyst of the fifth metacarpal of the right hand (myxochondroma).

HISTORY.—Fell on the hand, which became swollen (Nov. 20, 1923).

ON EXAMINATION (Nov. 27).—Swelling and thickening over the distal part of the fifth metacarpal; crepitus demonstrable.

X ray.—Shows fracture through a thin-walled cyst in the fifth metacarpal (*Fig. 19*).

TREATMENT (Nov. 30).—*Curettage,*



FIG. 20.—*Case 5.* Cyst wall with contents. Shows chondroma tissue.



FIG. 21.—*Case 5.* Two years later. Cyst completely obliterated by dense bone.

cauterization, bone-graft. The interior was filled with friable granulation-like tissue. No fibrous lining demonstrable.

HISTOLOGICAL DIAGNOSIS.—Myxochondroma (*Fig. 20*).

RESULT.—Two years later—cyst completely obliterated (*Fig. 21*).

Case 6.—C. F., female, age 16. (1) Cyst of the middle phalanx of the right index finger. (2) Cyst of the metacarpal (chondroma).

HISTORY.—Swelling of the right index finger noticed for seven years. No pain or trouble until the hand was hurt at work (Nov. 9, 1924).

ON EXAMINATION (Nov. 18).—Swelling and thickening of the middle phalanx of the index finger of the right hand. No crepitus.

X ray.—Shows clear-cut circular cyst, occupying the middle third of the middle phalanx; no trabeculation. Also cystic change in the distal half of the metacarpal of the index.

TREATMENT (Dec. 12).—*Curettage, cauterization, bone-graft.* (1) The cyst in the phalanx was found to be filled with closely packed glistening material. The bone shell showed no lining. (2) The cavity in the metacarpal contained soft tissue, white and glistening, mixed with loose vascular material. After complete evacuation of the contents of the bone cysts, small bone-grafts were inserted.

HISTOLOGICAL DIAGNOSIS.—Chondroma.

RESULT.—Four years later the patient writes to say the finger is quite normal. Final radiograph not available. A radiograph four months after the operation showed the cysts in process of obliteration.

Case 7.—L. W., female, age 13. Cyst of the fifth metacarpal of the left hand (chondroma).

HISTORY.—Contusion of left hand followed by swelling and pain (Feb. 14, 1925).

ON EXAMINATION (Feb. 17).—Swelling and tenderness with some thickening of the distal half of the fifth metacarpal.



FIG. 22.—*Case 7.* Cyst of the fifth metacarpal (chondroma). Shows multicystic expansion of metacarpal; clear-cut outline. Note: the cyst does not invade the epiphysis.



FIG. 23.—*Case 7.* Four years later. Cyst completely obliterated by bone; metacarpal is thickened.

X ray.—Shows cystic expansion of the metacarpal with coarse trabeculation. No sign of spontaneous fracture; cortex thin on ulnar border (*Fig. 22*).

TREATMENT (March 9).—*Curettage, cauterization, bone-graft*. Contents of cyst, semi-translucent sago-like material; no traces of fibrous lining.

HISTOLOGICAL DIAGNOSIS.—Chondroma.

RESULT.—Four years later—cyst completely obliterated (*Fig. 23*).

Case 8.—M. G., female, age 26. Cyst of the right middle metacarpal (chondroma).

HISTORY.—Injury to the right hand followed by pain and swelling (March 28, 1925).

ON EXAMINATION (April 7).—Swelling in the region of the head of the metacarpal of the middle finger of the right hand, with bony thickening.

X ray.—Shows cystic expansion of distal half of the third metacarpal with coarse trabeculation; fairly thick cortex.

TREATMENT (April 27).—*Curettage, cauterization, bone-graft*. Contents of cyst, sago-like material.

HISTOLOGICAL DIAGNOSIS.—Chondroma.

RESULT.—Four months later (patient not traced beyond this date)—cystic area almost completely obliterated by new bone.

Case 9.—E. E., female, age 22. Cyst of the proximal phalanx of the left index finger (myxochondroma).

HISTORY.—Swelling of the index finger noticed for four years, slowly increasing in size. Very little disability.

ON EXAMINATION (Jan. 10, 1928).—Slight thickening of the base of the proximal phalanx of the index finger.

X ray.—Shows cystic expansion of the base of the proximal phalanx of the index finger.

TREATMENT (Jan. 13).—*Curettage, cauterization, bone-graft*. Material evacuated from the cyst consisted of a mixture of cancellous bone and pale sago-like tissue.

HISTOLOGICAL DIAGNOSIS.—Myxochondroma.

RESULT.—One year and eight months later—trace of bone-graft showing; cyst almost completely obliterated.

Case 10.—V. W., female, age 16. Cyst of the proximal phalanx of the right little finger—spontaneous fracture (osteitis fibrosa).

HISTORY.—Little finger forcibly hyperextended by fall. This was followed by swelling and tenderness.

ON EXAMINATION (Jan. 13, 1928).—Swelling and thickening of proximal phalanx of the little finger.

X ray.—Shows fracture through a cyst (*Fig. 24*).

TREATMENT (March 26).—*Curettage, cauterization, bone-graft*. Contents of cyst, mixture of tough fibrous tissue and cancellous bone; tags of lining.

HISTOLOGICAL DIAGNOSIS.—Osteitis fibrosa (*Figs. 25, 26*).

RESULT.—Fifteen months later—cyst obliterated by dense bone (*Fig. 27*).



FIG. 24.—*Case 10*. Cyst of the proximal phalanx of the little finger (osteitis fibrosa); cyst occupying middle three-fifths of phalanx with fracture through the middle. Note: the cyst does not invade the old epiphysis.



FIG. 25.—Case 10. Cyst wall and contents. Bone trabeculae undergoing erosion; connective-tissue formation.



FIG. 26.—Case 10. Cyst contents. A giant-cell tumour area.

Case 11.—R. A. B., male, age 45. Cyst of the fifth metacarpal of the right hand (osteitis fibrosa).

HISTORY.—For the past three years swelling noticed of the right hand over the knuckle-joint of the little finger. No history of trauma.

ON EXAMINATION (Feb. 25, 1926).—There is a small elastic swelling over the dorsum of the metacarpo-phalangeal joint of the little finger which does not appear to be connected with the tendon sheath or directly with the joint. Movements of the metacarpo-phalangeal joint perfectly free.

X ray.—Shows very slight expansion of the head of the fifth metacarpal, with a cystic change in the interior. The cyst is circumscribed and there is no trabeculation.

TREATMENT (March 2).—*Removal of extra-osseous tumour; curettage of bone cyst.* A soft fleshy tumour composed of greyish-red tissue, gelatinous and friable, was found embracing the neck of the metacarpal bone. The tissue was continuous with similar material in the interior of the metacarpal neck through a small perforation on the ulnar aspect. One small mass of the intracystic tissue was dark red in colour and of firm consistency. The extra-osseous tumour was removed and the bone cyst curetted and cauterized with pure carbolic acid in the usual way.

HISTOLOGICAL DIAGNOSIS.—Osteitis fibrosa.

RESULT.—Three and a half years later (1929)—no recurrence. Final X-ray appearances not available.

COMMENT.—Histological interpretation difficult.

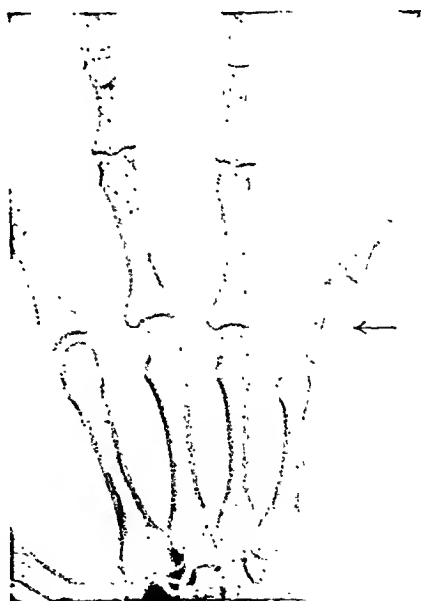


FIG. 27.—Case 10. Fifteen months later. Cyst obliterated by dense bone.

Case 12.—C. B., male, age 29. Cyst of the first metatarsal of the left foot—spontaneous fracture (osteitis fibrosa).

HISTORY.—Swelling and pain followed dropping of a plank on the left foot (Jan. 3, 1929).

ON EXAMINATION (March 31).—Swelling and thickening of the distal part of the first metatarsal.

X ray.—Shows cystic expansion of the middle two-thirds of the first metatarsal (Fig. 28).

TREATMENT (April 5).—Curettage, cauterization, bone-graft. Contents of the cyst consisted of sago-like material mixed with a certain amount of fibrous tissue and cancellous bone. No cyst lining present.

HISTOLOGICAL DIAGNOSIS.—Osteitis fibrosa.

RESULT.—Nine months later—metatarsal thickened. Radiograph shows obliteration of cyst proceeding.



FIG. 28.—*Case 12.* Cyst of the first metatarsal (osteitis fibrosa). Cystic expansion of the metatarsal; outline clear-cut; epiphysis not invaded. Fracture through cyst wall.

Group II.—NO HISTOLOGICAL DIAGNOSIS AVAILABLE.

Case 13.—H. G., male, age 37. (1) Cyst of the fourth metacarpal of the left hand. (2) Cyst of the proximal phalanx of the fourth finger.

HISTORY.—The left hand was jammed between the handle and the side of a moulding box (Feb. 28, 1925).

ON EXAMINATION (April 22).—Slight irregularity and tenderness in the region of the neck of the fourth metacarpal. No interference with mobility.

X ray.—Shows: (1) Multicystic expansion of the neck of the fourth metacarpal; (2) Early cystic expansion of the base of the proximal phalanx (fourth).

TREATMENT.—Exploration of the cyst advised, but not accepted by patient.

RESULT.—Unknown.

Case 14.—J. B., male, age 48. Cyst of the proximal phalanx of the left index finger (fracture).

HISTORY.—The patient struck the left index finger with a hammer (Oct. 15, 1925).

ON EXAMINATION (Nov. 16).—Tenderness over the proximal phalanx and crepitus elicited. Some limitation of movement at the metacarpo-phalangeal joint.

X ray.—Shows small cyst in the base of the first phalanx, with crack extending from the medial side of the base to cyst.

TREATMENT.—Fixation on splint. At the end of a fortnight the tenderness had disappeared and the patient returned to work.

RESULT.—Four years later—slight thickening of the proximal phalanx. No limitation of movement at the metacarpo-phalangeal joint. Radiograph shows little if any change in size or structure of the cyst.

COMMENT.—Probably a quiescent or healed lesion.

Case 15.—J. B., male, age 27. Cyst of the third metatarsal of the right foot (fracture).

HISTORY.—A cylinder of gas fell on the dorsum of the patient's foot whilst at work. Three months previously he had had a similar accident, but without any fracture.

ON EXAMINATION (Nov. 18, 1927).—Pain and slight swelling in region of the head of the third metatarsal.

X ray.—Shows small thick-walled cyst in the head of the third metatarsal with pathological fracture.

TREATMENT.—Strapping of the foot. Three months later the patient returned to work, but still complained of occasional pain over the site of the fracture. Operation was considered, but for economic reasons was deferred.

RESULT.—A year later—in a radiogram the cyst shows no change in size or structure.

COMMENT.—Probably a quiescent or healed lesion.

Case 16.—E. G., female, age 32. Cyst of the middle phalanx of the left ring finger (pathological fracture).

HISTORY.—Patient fell heavily on the left hand (May 28, 1927).

ON EXAMINATION (May 31).—Tenderness and swelling around the first interphalangeal joints of the left ring and little finger.

X ray.—Shows very small cyst eccentrically placed in the base of the middle phalanx of the left ring finger, with pathological fracture.

TREATMENT.—Splintage. The patient disappeared after a few attendances.

RESULT.—Two and a half years later—no trace of disability. The cyst shows unchanged in a radiogram.

Case 17.—F. U., male, age 11. Cyst of the proximal phalanx of the left little finger (fracture).

HISTORY.—Patient received a kick on the little finger (Oct. 2, 1928).

ON EXAMINATION (Oct. 9).—Thickening and tenderness over the proximal end of the first phalanx of the little finger.

X ray.—Shows small cyst in the base of the phalanx.

TREATMENT.—Temporary fixation on a splint. Patient did not report again.

RESULT.—Not known.

ANALYSIS OF THE CASES.

Number of patients: 8 males, 9 females. Ages: under 20, 8; 20-30, 4; 30 and over, 5.

Number of cysts: 20, of which 13 were explored—curettage and cauterization, 3; curettage, cauterization, and bone-graft, 10.

Digits affected:—

Little finger	9	Thumb	1
Ring	1	Great toe	1
Middle	1	Third toe	1
Index	3			

Bones affected: Phalanges—proximal, 9, middle or distal, 2; metacarpals, 7; metatarsals, 2.

CLINICAL AND RADIOGRAPHIC SIGNS.

Symptomatology.—The 17 cases in the writer's series fall into two main clinical groups: (1) The first is made up of cysts discovered by radiographic examination soon after the occurrence of a localized injury (13 cases). Eight of these showed evidence of fracture through the cyst wall in the radiogram, but unmistakable clinical signs of fracture were rarely demonstrated. (2) In the second or minority group there is a history of a gradual onset of pain, localized swelling, or the appearance of an actual lump (4 cases). In *Case 4* the lump—a solid chondroma—was attached to the fifth metacarpal, but the cyst occupied the proximal phalanx of the same digit and gave no signs of its existence until revealed in a radiogram. The cyst was also discovered accidentally in *Case 11* owing to the presence of a soft extra-osseous swelling.

In the 16 cysts of the Johns Hopkins series already quoted, the clinical signs, which are briefly tabulated, are in contrast with the above; for in 3

only is trauma recorded as the incident which presumably led to the discovery of the cyst.

Skeletal Site.—The preponderance of the upper extremity is illustrated in the figures of both series. The digit most commonly affected appears to be the *little finger* (9 out of 17 patients), with the *index* finger next in order of frequency. In my own series the phalanges outnumber the metacarpals (11 phalangeal; 7 metacarpal). The majority of phalangeal cysts are found in the proximal phalanx; the middle phalanx is an uncommon site; and the distal phalanx appears to be immune. In the Johns Hopkins series the metacarpals are more frequently involved:—

Hand	{ Metacarpal .. 7	Foot	{ Metatarsal .. 4
	{ Phalanx .. 4		{ Phalanx .. 1

Taking the two series together, however, the incidence in the phalanges is greater.

Sex and Age.—The sexes appear to be almost equally liable. The age incidence in the two series combined is as follows:—

Under 20	12
20-30	7
30 and over..	..	11

These figures refer to the age when the cyst was discovered. In a number of the older patients it is evident that the lesion had been in existence for a long period.

Radiographic Signs.—It is unnecessary to describe in full detail the ordinary radiographic appearances, which are comparable in every way to those so often depicted in the more familiar cysts of the major long bones at various stages in their life-history. There are, however, certain features relating to the site of origin, mode of extension, and healing of the miniature cysts which call for comment. It is probable that the lesions originate in the growing end (metaphysis) of the affected metacarpal or phalanx, although when discovered, even in a younger patient, the cystic area has usually been pushed down along the interior of the shaft by growth in length. This is, of course, a recognized phenomenon in the solitary cyst (osteitis fibrosa) of the long bones. The integrity of the cancellous tissue of the old epiphysis is also well demonstrated in the adult patients (*see Figs. 19 and 28*). These distinctions are significant, for it is well established that the bone cyst of osteitis fibrosa develops in the *metaphysis*, and the giant-cell tumour (myeloid sarcoma) in the old *epiphysis* (Bloodgood). The chondroma of the long bones is also a metaphyseal lesion—a fact illustrated best of all in the condition known as multiple cartilaginous exostoses (diaphyseal aclasis). Thus on radiographic evidence alone, the above series would be unlikely to contain a pure giant-cell tumour cyst—a presumption confirmed by the histological findings.

In a few of the adult patients the radiographic appearances suggest that spontaneous arrest or actual healing of the lesion has occurred. This is obviously true in *Case 14*, in which a radiogram four years later shows the cyst practically unchanged. Further reference will be made to this observation when the treatment of the cysts is discussed.

PATHOGENESIS.

In the group of cysts in my own series in which a histological examination was made of material obtained at operation, two distinct types of lesion were identified: (1) Chondroma (myxochondroma), 8 cysts; (2) Osteitis fibrosa (5 cysts).

1. **Chondroma (Myxochondroma).**—Chondromatous cysts have been recognized by pathologists and surgeons for many years, and most of the earlier recorded metacarpal and phalangeal cysts belong to this category. When such a cyst is opened the naked-eye appearance of the interior is characteristic. Usually the cavity is completely filled with a white glistening material not unlike sago, mixed with friable granulation-like tissue and soft cancellous bone. No fibrous lining is demonstrable. In the histological picture the most interesting feature is the presence of areas showing stellate cells sometimes hardly distinguishable from the cells of a pure myxoma (*see Fig. 15*). The latter bone tumour, the existence of which has been doubted by many, is regarded by Bloodgood as a true pathological entity, with a predilection for the phalanges. Two examples are included in his eight phalangeal cysts reported in 1920, and reference is made to two similar cases previously recorded by Codman and Rhodes. In my series the myxomatous areas form a small part only of the tumour tissue, which is predominantly cartilaginous in structure. For this reason the lesions have been classified as chondroma or myxochondroma.

2. **Osteitis Fibrosa.**—Until recent years it was generally taught that non-tumour lesions were practically unknown in the miniature bone cysts. This belief is illustrated in the earlier experience of Bloodgood,⁴ who up to 1920 could find no record of an osteitis fibrosa cyst affecting the phalanges, although he had seen a few examples in the metacarpals and metatarsals.

In five cysts in my series the naked-eye appearances of the cavity suggested a diagnosis of osteitis fibrosa, which was confirmed by a histological examination of the lining and contents. These cysts are usually found to be incompletely filled by tissue, and in the later stages may be entirely empty. The tough fibrous lining which peels easily, and the clumps of similar tissue in the interior, serve to distinguish this lesion from the typical myxochondroma. But in both varieties of cyst the admixture of cancellous bone and granulation-like areas may mask the specific tissue.

The histology of osteitis fibrosa in its various phases is now familiar to the surgical pathologist—namely: the early resorption of bone; the spindle-cell reaction which leads to fibrous tissue substitution of bone; the formation of islands of osteoid bone; recent hæmorrhages in close relation to areas showing the cellular picture of the giant-cell tumour. (*See Figs. 12, 25, 26.*) All these changes are usually demonstrable in the same cyst in different parts of the wall and contents. In recent years our knowledge of the histogenesis of osteitis fibrosa and its relation to the giant-cell tumour has been particularly enriched by the writings of Barrie,⁸ Stewart,⁹ the later work of von Recklinghausen,¹⁰ Dawson and Struthers,¹¹ and Bloodgood¹² and his school. A detailed exposition of this most fascinating chapter in bone pathology lies outside the scope of this paper. Reference, however, must be made to the

somewhat atypical histological findings in the miniature bone cysts of the Johns Hopkins collection of 175 bone cysts and 226 giant-cell tumours. As a result of a careful re-examination of this material, Geschickter and Copeland⁷ have recognized certain transitional lesions which form a link between osteitis fibrosa and the giant-cell tumour. These lesions conform to two histological types: (1) The first, which is described as a *giant-cell variant of the bone cyst*, represents the uniform histological picture found in 16 cysts of the long bones and 17 cysts of the small bones and flat bones (8 cysts of the digital bones). The distinguishing mark of this lesion is the presence of giant cells smaller than the myeloid cell sparsely scattered in a stroma consisting mainly of fibroblasts and containing bone islands. (2) The second, described as a *spindle-cell variant of the giant-cell tumour*, was discovered in 20 specimens (8 cysts of the digital bones). In these cysts the characteristic myeloid giant cell was present in large numbers, but unlike the giant-cell tumour proper, the spindle-cell and fibrous-tissue formation predominated in the stroma. In the 16 digital bone cysts the two transitional lesions were represented in equal numbers, and it is a striking fact that no single cyst in this group showed the conventional histology of osteitis fibrosa or giant-cell tumour. We must therefore assume that Geschickter and Copeland hold the opinion that the fundamental histological picture in the digital bone cyst (excluding the chondroma group) is a hybrid lesion. It is interesting to note that in the specimens examined by these writers, 8 cysts had been obtained by amputation of the affected digit, and 5 by resection or excision of the affected bone. Such material afforded ample opportunity for a complete topographical survey of the histogenesis of these comparatively tiny cysts. For such a purpose, my own scanty material derived solely from the curettage of five cysts was in no way comparable. A further scrutiny of the microscopic preparations in my series fails to disclose any evidence which might be used either to support or challenge the findings of Geschickter and Copeland. In one cyst only (*Case 10*) was a small giant-cell tumour area discovered (*see Fig. 26*). In this the giant cells are few in number and are smaller than the classical myeloid cell.

The accuracy of the histological observations in the Johns Hopkins series is beyond criticism, but the interpretation is not so readily acceptable. In my judgement the creation of two distinct histological entities tends to disturb our present simple conception of the histogenesis of osteitis fibrosa, in which, despite its kaleidoscopic character, the meaning of the different patterns is clear. Furthermore, if I read the authors correctly, their main thesis is concerned as much with the essential contrast between the anatomical and clinical pictures of the bone cyst and the giant-cell tumour as with the underlying unity of the minute anatomy of these lesions. For special emphasis is laid on the fact that the *bone cyst* is a lesion of early youth (age 10-15), which originates in the metaphyseal cancellous tissue, the favourite sites being the upper end of the humerus, the upper end of the femur (trochanteric region), and the upper end of the tibia. The clinical sequence of events is: trauma—spontaneous fracture (45 per cent of all cases)—discovery of the cyst. The *giant-cell tumour*, on the other hand, is a lesion of adult life (age 20-30), originating in the subcortical cancellous tissue of the epiphysis, the

favourite sites being the lower end of the femur, the upper end of the tibia, and the lower end of the radius. The clinical sequence is: trauma—pain—tumour, with fracture as an occasional late phenomenon (14 per cent of all cases). Whether, as held by Geschickter and Copeland, osteitis fibrosa is merely a non-specific tissue reaction, or, as held by Dawson and Struthers,¹¹ a true pathological entity, in the bone cyst of young people this lesion is pre-eminently a repair or healing process. But the giant-cell tumour proper is an invasive and destructive lesion, a true neoplasm, although admittedly benign.

For the purpose of surgical teaching, I believe that we should do well to continue to emphasize these two contrasted pictures, which form a sound guide to the prognosis and treatment of the lesions under discussion. At the same time, however, it must be realized that the two lesions have a common histological denominator—the giant-cell area. This fact, known since von Recklinghausen's original contribution, has repeatedly led to confusion in diagnosis.

In the giant-cell areas so often discovered in solitary bone cysts and in non-cystic osteitis fibrosa, the cellular picture is by no means constant. Thus one area may reproduce the histology of the typical giant-cell tumour—that is, large multinucleated giant cells in abundance, embedded in a round-celled stroma; another area may show a different type of giant cell or a more fibrous stroma. These variations correspond to the hybrid lesions described by Geschickter and Copeland. Now if from such variations two histological entities are to be separated, and each derived from a different basic lesion, we should expect to find one type of hybrid cyst approximating in its clinical and anatomical features to osteitis fibrosa, and the other to the true giant-cell tumour. As far as I am able to judge, there seem to be no such outstanding differences between the two histological groups which comprise the Johns Hopkins series of 16 miniature long bone cysts. For this reason it would be more logical to regard these cysts as belonging to a single anatomical, clinical, and pathological group, in which are exhibited two different phases of the well-known composite histological picture of osteitis fibrosa.

When the debatable hybrid lesions are excluded, the pure giant-cell tumour appears to be a rare tumour of the digital bones. There is no single example in the 144 *typical* giant-cell tumours in the Johns Hopkins material, nor in a series of 258 similar tumours recorded by Christensen.¹³ The following case in my own collection, on radiographic evidence was at first grouped with the metacarpal cysts, but later was rejected when the tumour was proved to be a solid growth replacing the metacarpal.

Case 18.—M. A., female, age 35. Giant-cell tumour (myeloid sarcoma) of the metacarpal of the left thumb.

HISTORY.—Swelling of the left thumb developed without known reason. No record of injury. Eight months' duration.

ON EXAMINATION (Nov. 12, 1928).—Swelling of the thumb involving the whole length of the metacarpal bone. Skin shows distended veins.

X ray.—Shows the metacarpal represented by a bone shell with a pseudo-cystic appearance; interior is occupied by trabeculae and tumour tissue, which is radiopaque (*Fig. 29*).

TREATMENT (Nov. 16, 1928).—*Resection of thumb metacarpal with tumour; replacement by bone-graft.* The swelling was explored and found to consist of solid tumour

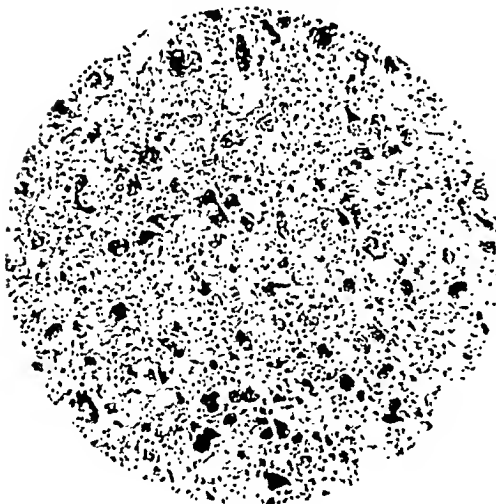
tissue, dark red in colour. Owing to the almost complete disappearance of the bone shell, it was found impossible to carry out the usual curettage and cauterization. The whole metacarpal and tumour mass was excised, leaving the distal surface only.



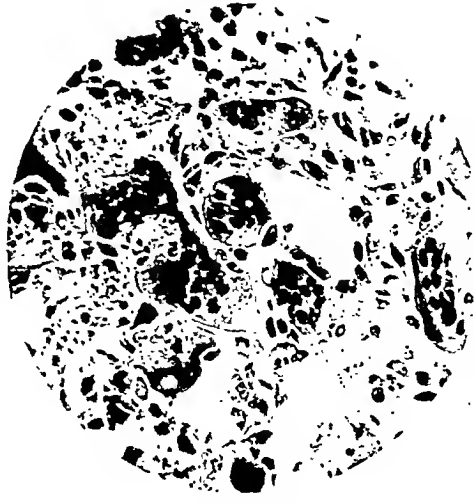
FIG. 29. Case 18. Giant-cell tumour of the first metacarpal. Expansion of remains of metacarpal, which has been almost replaced by tumour; trabeculation marked.

After cauterization of the bed a tibial bone-graft was impacted into the trapezium and into the cancellous tissue of the remains of the metacarpal head.

HISTOLOGICAL DIAGNOSIS.—Giant-cell tumour (Figs. 30, 31).



Low power.



High power.

FIGS. 30, 31.—Case 18. Typical giant-cell tumour.

DIAGNOSIS.

Discovery of the Cyst.—As the majority of cysts remain for some time almost silent in a clinical sense, a diagnosis in the early stages is impossible without the aid of a radiogram. A cyst may reasonably be suspected when a painless localized thickening of the base of a proximal phalanx or of the neck of a metacarpal—particularly in the little finger—has been known to exist for a considerable time. The presence of a solid tumour, or a soft swelling without any inflammatory signs, attached to a metacarpal or a phalanx, is also suggestive.

Differential Diagnosis Between Chondroma and Osteitis Fibrosa.—When the cyst has been discovered the problem is one of distinguishing between the two classical lesions, chondroma and osteitis fibrosa. If the patient is a very young child, or if a solid tumour is attached to the same digit, the lesion is almost certain to be chondroma. But in the older child or adult, as far as my limited observations go, there appear to be no convincing points of distinction between the two varieties of cyst. The final proof will thus depend on a histological examination of material obtained from the interior of the cyst.

Alternative Lesions.—We must consider the remote possibility of the occurrence of lesions other than those which are known to exist in the majority of the miniature bone cysts:—

Neoplasms.—Reference has already been made to the rarity of the giant-cell tumour in the digital bones. Malignant tumours, whether osteogenic sarcoma or secondary carcinoma, are also extremely rare, and in the phalanges are unknown. A radiogram of an osteogenic sarcoma of the first metatarsal is reproduced by Kolodny¹⁴ in his well-known monograph, but the appearances are unlike those of an actual bone cyst.

Subacute Inflammatory Lesions (daetylitis—tubercle or syphilis; pyogenic osteitis).—No difficulty should be experienced in excluding such conditions under ordinary circumstances; but when the inflammatory signs are trivial, and the radiogram shows an early localized expansion of a phalanx or metacarpal, the diagnosis may be temporarily in doubt.

TREATMENT.

Owing to the lack of accurate knowledge bearing on diagnosis and prognosis, a considerable proportion of metacarpal and phalangeal cysts in the past have been treated by operation. The Johns Hopkins series, consisting for the most part of specimens collected from different sources over a period of years, illustrates the uncertainty which has determined the variety of procedures adopted. Thus 8 cysts were treated by amputation of the digit, 5 by resection or excision of the cyst or bone, 2 by curettage, and 1 by X rays. In the earlier cases in my own series the cysts were explored as a routine, for three reasons: (1) To establish a diagnosis; (2) Because it seemed impossible to forecast whether a particular lesion was likely to progress or become arrested; and (3) Because the experience of operating on solitary cysts of the larger bones had shown that rapid obliteration of the cystic area usually followed the operation of curettage and the insertion of a bone-graft.

With the fuller knowledge now available, we are able to define the rôle of operative treatment with more authority.

Indications for Operation.—There is evidence that a *chondromatous* cyst which has come to occupy a considerable area in a metacarpal or phalanx may shrink and ultimately acquire a thick wall. In the not uncommon multiple solid chondromata of the digits, which are often associated with widespread cystic or pseudo-cystic changes in several bones, considerable bony reconstruction is known to occur. But it is doubtful whether complete spontaneous obliteration ever takes place in the solitary chondromatous cyst. In contrast with this capacity for healing, we must also reckon with the invasive characteristics of certain types of myxochondroma. Bloodgood¹⁵ has always looked with extreme suspicion on this tumour, which in his experience resembles the pure myxoma in its liability to recur locally after incomplete curettage.

The pronounced healing capacity of the average *osteitis fibrosa* cyst in younger individuals is admitted by all experienced surgeons. Natural repair is often hastened by the occurrence of spontaneous fracture, but a second fracture is occasionally seen at a later date where the healing proceeds sluggishly. In the adult, however, a solitary lesion may show a tendency to progress as in generalized osteitis fibrosa.

With a due regard to these considerations, the indications for operation may be stated as follows: A cyst discovered accidentally which shows signs of quiescence—for example, a thick wall—should be treated conservatively, and its condition observed in radiograms taken at regular intervals. But cysts in process of extension in which the shell is thin, and especially at the time of fracture, should be explored. This is advisable even in younger patients, and more especially in the adult, in whom both the myxochondroma and osteitis fibrosa are liable to be destructive.

Operative Technique.—There is ample proof that these miniature cysts can be effectively treated by the simple operation of curettage of the contents combined with cauterization of the shell by pure carbolic acid. This procedure, introduced by Bloodgood long ago for the treatment of giant-cell tumour, has been consistently used in my own clinic for all types of benign bone cyst. In addition, it has been my practice to stimulate the obliteration of the cyst by filling the cavity as completely as possible with one or more autogenous grafts. With few exceptions, the operation of curettage and cauterization combined with a bone-graft has been employed in 13 cysts of the series recorded in this paper, and its success leads me to regard it as the essential method of treating these lesions. The mutilating procedures of resection or amputation should be reserved for those very rare neglected cysts in which the lesion after perforating the bone shell has spread beyond its confines.

SUMMARY.

1. The clinical and radiographic pictures of a series of 20 cysts of the long bones of the hand and foot occurring in 17 patients are analysed and compared with those of similar cysts recorded in the literature.

2. The bones involved in order of frequency are the *phalanges* (of the hands), *metacarpals*, and more rarely, the metatarsals. The cysts originate in the growing ends (metaphysis). The favourite digit is the little finger.

3. The majority of cysts develop insidiously, remain latent for a time, and are discovered after the occurrence of local injury. Spontaneous fracture is a fairly common phenomenon.

4. In 13 cysts in which a microscopie examination was made of material removed at operation, two varieties of lesion were distinguished: (a) Chondroma (myxochondroma); (b) Osteitis fibrosa. For practical purposes these two lesions comprise the whole morbid histology of the miniature bone cysts. Alternative lesions, such as giant-cell tumour (myeloid sarcoma) or malignant tumours, are almost unknown. The differential diagnosis between the two standard lesions is usually impossible on clinical and radiographic evidence alone.

5. In both types of cyst spontaneous arrest or healing may occur, particularly in young patients. For such cysts no form of operative treatment is required.

6. Cysts which are actively extending, or where the bone shell is perforated by fracture, should be explored.

7. The most effective method of eradicating the lesion is to curette the contents and cauterize the interior of the cyst with pure carbolic acid. This procedure is best combined with the insertion of one or more autogenous bone-grafts, which hasten the obliteration of the cystic area.

REFERENCES.

- ¹ VON RECKLINGHAUSEN, *Festsch. f. Virchow*, 1891.
- ² JONES, R., and MORGAN, D., *Arch. of Roentgen Ray*, 1907, April-May.
- ³ ELSLIE, R. C., *Brit. Jour. Surg.*, 1914, ii, 17.
- ⁴ BLOODGOOD, J. C., *Amer. Jour. Orthop. Surg.*, 1920, 597.
- ⁵ SILVER, D., *Ibid.*, 1912, 563.
- ⁶ ROEDERER, M. C., *Rev. d'Orthop.*, 1923, Nov., x, 6.
- ⁷ GESCHICKTER, C. F., and COPELAND, M. M., *Arch. of Surg.*, 1929, Aug., xix, 2.
- ⁸ BARRIE, G., *Jour. Bone and Joint Surg.*, 1922, Oct., iv, 4.
- ⁹ STEWART, M. J., *Lancet*, 1922, ii, 1106.
- ¹⁰ VON RECKLINGHAUSEN, *Untersuchungen über Rachitis und Osteomalacia*, 1910.
- ¹¹ DAWSON, J. W., and STRUTHERS, J. W., *Edin. Med. Jour.*, 1923, Oct., xxx, 10.
- ¹² BLOODGOOD, J. C., *Ann. of Surg.*, 1910, lii, 145; 1912, lvi, 210; 1919, lxi, 305; *Jour. Bone and Joint Surg.*, 1921, xviii, 8.
- ¹³ CHRISTENSEN, F. C., *Ann. of Surg.*, 1925, lxxxi, 1074.
- ¹⁴ KOLODNY, A., *Bone Sarcoma*, 1927, Chicago.
- ¹⁵ BLOODGOOD, J. C., *Ann. of Surg.*, 1920, lxxii, 712.

GAS GANGRENE IN CIVIL SURGERY.

BY NILS L. ECKHOFF,

SURGICAL REGISTRAR AT GUY'S HOSPITAL, LONDON.

For the purposes of this review all cases of gas gangrene occurring at Guy's Hospital in the six years 1924-9 are included, and most of these have been personally observed by the writer. To lay stress upon the occurrence of gas infection complicating 'clean' operations is the main object of this paper.

The total number of cases occurring during the period is 23, and they may be at once divided into those occurring soon after and as a direct result of gross wound contamination, and those following 'clean' operations—or, more shortly, into accident and non-accident cases. The former will be briefly described first.

ACCIDENT CASES.

The 12 accident cases were as follows:—

	CASES
Compound fracture of tibia and fibula ..	1, 3, 9
" " " femur ..	2
" " " patella ..	5, 6
" " " tibia, fibula, ulna and humerus ..	8
Lacerated wound of leg ..	4, 12
" " " and abdominal wall ..	7
" " " hand ..	11
Incised wound of arm ..	10

Case 1.—H. W. P., male, age 17. Admitted on March 14, 1924, for compound fracture of the tibia and fibula of the right leg following a lift accident.

OPERATION (March 14, 6.0 p.m.).—Excision of wound edges. Fragments manipulated into position. Wound sutured.

After operation: pulse 82, respirations 20, temperature 98.8°. At end of thirty hours obvious gas infection of wound had set in; temperature 101.6°, pulse 144 (having risen from 80 in six hours).

March 15.—12.0 p.m.: Amputation through mid third of thigh. Antitetanic serum 1500 units. Anti-gas-gangrene serum 50 c.c.

March 16.—A.G.G.S. 50 c.c.

March 18.—A.G.G.S. 50 c.c.

March 21.—Much pain in left arm.

March 23.—Subperiosteal abscess of left humerus opened. *Streptococcus longus* cultivated.

March 28.—Subperiosteal abscess of left ulna opened. *Str. longus* cultivated. Stump looking well.

April 3.—Arm again explored through further incision. Much pus.

April 8.—A.T.S. 50 c.c., following which there was marked anaphylactic shock.

April 12.—Death.

Hectic temperature persisted throughout illness.

Comment.—This patient was unfortunate to succumb to a streptococcal pyæmia after having satisfactorily recovered following the amputation for

gas gangrene. Anaphylactic shock appearing after the fourth injection of serum was also interesting.

Case 2.—L. C., male, age 16. Admitted on July 29, 1924, for compound fracture of the left femur with severe lacerations of the skin, following a street accident.

OPERATION (July 29, 10.0 p.m.).—Amputation through subtrochanteric region of the femur, with flaps and drainage.

After operation : pulse 140, temperature 100°, respirations 26. A gum infusion of 1½ pints was administered to help combat the loss of blood : 1500 units of A.T.S. and 10 c.c. A.G.G.S. injected intramuscularly.

July 30.—10.0 a.m. : One pint of saline intravenously. 10.0 p.m. : Temperature 102° (axilla), pulse 130–140.

July 31.—2.0 a.m. : Gas gangrene developed in stump, spreading up to abdominal wall. Pulse 170, temperature 103° (groin). Patient very much collapsed. Wound re-opened. Infected muscles excised as far as possible, up to pelvic bones. A.G.G.S., 80 c.c., injected into leg and abdominal wall. Flavine dressing. *B. coli* and *B. welchii* cultivated.

9.0 p.m. : Temperature 101°, pulse 160, respirations 25. Defibrinated blood transfusion, 16 oz., given. Severe reaction with rigor (temperature 106° and pulse-rate of 180).

Aug. 1.—Temperature 98.4°, pulse 110, respirations 22. Remarkable improvement in general condition. Recovery was steady from this date, marred only by the necessity of a few minor operations for the repairing of the wound.

Oct. 15.—Discharged with stump well healed. Seen lately (1929) in very satisfactory condition.

Comment.—An extremely ill boy suffering in the first place from exsanguination ; later developed gas gangrene in a part where free surgical removal was out of the question. Responded satisfactorily to serum treatment and flavine dressings to the opened wound. Remarkable recovery following transfusion, though the immediate reaction was intense. This phenomenon has been seen several times.

Case 3.—D. C., female, age 22. Admitted on July 6, 1924, for fracture of the left tibia and fibula produced by a piano falling on the leg. Grazing of skin and one minute punctured wound over the fibula. Leg placed on splint.

July 8.—Manipulation under anæsthetic. Retention of reduced fragments impossible owing to effusion and state of skin. Splint re-applied. Temperature 101°, pulse 100, respirations 20.

July 9.—6.0 p.m. : Intense pain in leg. Temperature 102°, pulse 130, thready (having risen steadily from 90 in eight hours), respirations 26.

Careful examination of leg revealed fine crepitation on outer side. Skin tense and shiny. Tympanitic note. Amputation performed above knee. Serum 30 c.c. (Leg showed typical changes of gas infection—muscles being red to grey—full of gas of putrid odour.) Cultivation *B. welchii*.

July 10.—Condition remarkably good and there was no pain. Pulse 100, temperature 101°. A.G.G.S. 40 c.c.

July 24.—Wound well healed.

Aug. 15.—Discharged fit.

Case 4.—C. S., male, age 28. Admitted at 2.0 p.m. on Sept. 6, 1924, for severe lacerations of the right leg. Exsanguination and shock. No fracture. Given axillary saline, 1 pint ; transfusion, 1 pint ; rectal saline, 1 pint, in the first twelve hours.

Sept. 7.—2.0 p.m. : Subtrochanteric amputation through femur. Suture and drainage. A.G.G.S. 40 c.c. Axillary saline 3 pints. Gum acacia 1 pint.

Sept. 8.—Pulse 160, temperature 101°, respirations 25. Gas infection of wound marked. Stitches removed. Gas serum 70 c.c. into stump muscles. Cultivation *Staph. albus*, *B. coli*, and *B. welchii*.

Oct. 17.—Stump of femur projecting through wound. Disarticulation at hip. Prolonged hectic temperature 100°–102°. Antistreptococcal serum, in two doses of 40 c.c., given a month later, appeared to bring about improvement.

March 8, 1925.—Discharged well.

Case 5.—E. Y., female, age 30. Admitted on Nov. 1, 1928, for severe lacerations of the right leg opening into the knee-joint, with fractured patella and fractured femoral condyle, following street accident.

OPERATION (Nov. 1, 10.0 p.m.).—Wound edges excised, bony fragments replaced. Wound sutured, with four rubber drains inserted. A.T.S. 1500 units.

Nov. 4.—Much pain. Temperature persistently high since operation, 101°–102°; pulse 110–126.

Nov. 5.—Wound looking well, but much pain.

Nov. 6.—Considerable exudation of pus from lower drainage wound. lower and outer flap of skin becoming gangrenous. Gas present in subcutaneous tissues—offensive odour. Patient was very collapsed. Pulse 130, temperature 104°.

7.15 p.m.: Amputation performed through upper third of thigh, affected muscles being excised widely. Wound unsutured—packed with gauze soaked in potassium permanganate. Carrel-Dakin tubes inserted. A.G.G.S. 40 c.c. intravenously, 20 c.c. locally. 11.0 p.m., transfusion.

Nov. 7.—12.45 a.m.: Death.

Comment.—One cannot help feeling that in this case the persistent high temperature and pulse, and extreme pain in the limb, ought to have promoted an earlier discovery of the gas infection.

Case 6.—E. W., male, age 46. Admitted at 6.0 p.m. on May 7, 1926, for compound fracture of patella and severely lacerated leg, following street accident, exposing the knee-joint and all the structures in the popliteal fossa.

OPERATION (May 7, 10.0 p.m.).—Excision of skin edges and all damaged tissue and cleansing with antiseptic solutions—drainage provided. No important vessels or nerves were damaged, but patella was missing. Wound edges drawn together; flavine dressing. A.G.G.S. 10 c.c. A.T.S. 1500 units.

May 8.—10.0 a.m.: Limb looking well. 10.0 p.m.: Impending gangrene. Limb cold, pulse not detected, skin blue and mottled. Temperature 100°, pulse 100.

May 9.—Limb obviously gangrenous. Putrid odour apparent. Temperature 101°, pulse 120. Patient collapsed. Operation 4.0 p.m.: Amputation through middle of thigh. A.G.G.S. 30 c.c. (Much gas, with putrid odour, was found in the leg. *B. welchii* was grown in the culture from the leg.)

May 10.—6.0 a.m.: Death.

Case 7.—W. W., male, age 65. Admitted at 2.0 p.m. on June 29, 1926, for laceration of lower abdominal wall and left thigh, following street accident.

6.0 p.m.: Grossly infected wound cleaned out, edges excised, and a few sutures inserted, after placing Carrel-Dakin tubes under the stripped-up tissues. A.T.S. 1500 units.

Pulse rose steadily from 80 to 110 during the day, whilst the temperature fell to 96°. Great collapse. Gas noticed in wound.

June 30.—2.0 a.m.: Wound opened up extensively and bathed in peroxide of hydrogen. 4.30 a.m.: Death.

Case 8.—A. L., female, age 46. Admitted at 10.0 p.m. on Nov. 17, 1926, for compound fracture of the humerus and olecranon, and compound fracture of the tibia and fibula, following street accident. Moderate shock.

OPERATION.—Immediate. Wound edges excised and sutured. There was no record of serum having been given.

Nov. 19.—Temperature 99.8°, pulse 120. Wound of arm was looking unpleasant.

Nov. 20.—Patient was very restless, with delirium tremens.

Nov. 23.—Leg showed subcutaneous ecchymosis, but was otherwise healthy. Arm œdematous, and skin about wound was dusky. Unpleasant odour. Temperature 102°, pulse 110.

Nov. 24.—Gas gangrene recognized in arm. Temperature 104°, pulse 110. Amputation through middle of humerus. A.G.G.S. 10 c.c. given.

Nov. 26.—Gas gangrene recognized in leg. Temperature 103°, pulse 140. Operation advised, but was refused.

Nov. 28.—5.20 p.m.: Death.

Comment.—A very bad subject, being alcoholic and having a history of past gonorrhœa and syphilis.

Case 9.—H. W., female, age 55. Admitted on Sept. 16, 1927, with a Pott's fracture (abduction fracture of the ankle of the third degree), not compound. Known to be a diabetic, but urinary incontinence made the recovery of urinary samples impossible at first. Later samples revealed strong reduction of Fehling's solution and a strongly positive ferric chloride test.

OPERATION.—The leg was manipulated and put in plaster, with division of the outer plaster case to facilitate subsequent rapid removal for inspection.

PROGRESS.—Remained comfortable for two or three days, when her general condition suggested early diabetic coma. Insulin was therefore exhibited.

Sept. 19.—The evening temperature rose to 102°, the pulse being steady at 120. Discoloration of the toes was noticed, and diabetic gangrene was suspected.

Sept. 20.—Pulse 120.

Sept. 21.—Crepitation discovered in the leg, and the diagnosis of gas gangrene made.

The general condition was very bad, but amputation was advised and carried out, above the knee, by the guillotine method. Flavine packs were applied, and gas-gangrene serum was injected locally. The patient died shortly after the operation. *B. welchii* grown in culture.

Case 10.—C. R., female, age 16. Admitted on Oct. 1, 1927, with an incised wound of the inner side of the left arm, just below the anterior axillary fold, received in a quarrel. On admission patient was extremely exsanguinated, and unconscious, with a flickering pulse at the right wrist of 140, and none at the left. Though capillary circulation at the finger-tips was just present, the left arm was cold and insensitive to stimuli.

OPERATION.—After resuscitation with warmth and gum and saline infusion, the wound was explored. The brachial artery and superior profunda were found completely divided, as also were the musculospiral, musculocutaneous, ulnar, and median nerves. The ends of the arteries were ligatured, and the ends of the nerves brought together and sutured after identification.

PROGRESS.—The pulse remained high, 130–150. The following day it rose steadily to 180, and the temperature rose to 104°.

Examination revealed that the whole arm below the wound was œdematous and tense, and showed marked subcutaneous emphysema. Patient moribund.

OPERATION.—After a blood transfusion of one pint (which produced some slight improvement) a guillotine amputation was performed just above the previous wound, with complete excision of the coracobrachialis and most of the triceps (which were heavily infected). Flavine packs, soaked in A.G.G.S., were laid on the raw surface of the wound. A further 60 c.c. of A.G.G.S. was injected into the thigh muscles.

Inspection of the amputated arm showed a violent infection with a gas-forming organism, later identified bacteriologically as *B. welchii*.

In spite of the critical condition before the operation, the patient made a steady recovery and was subsequently discharged on Dec. 13, with the stump satisfactorily healed.

Case 11.—W. H., male, age 50. Admitted on March 5, 1928, for acute gangrenous swelling of hand.

HISTORY.—Scratched hand three days before admission on a piece of wire, whilst hauling in fishing nets at sea. Hand became increasingly swollen, and later, black. Reported at Guy's Hospital as soon as ship touched port. Condition on admission: pulse 120, temperature 100°, respirations 30. Hand was intensely swollen below the wrist, with uniform black discoloration of skin, and many large bullæ containing blood-stained fluid. On palpation, crepitation could be made out. Œdema, but no fluctuation. Gas gangrene diagnosed.

OPERATION (March 5).—Amputation through middle of forearm. *B. welchii* cultured. A.G.G.S. 30 c.c. locally.

March 6.—A.G.G.S. 30 c.c. intramuscularly.

Recovery rapid and complete.

Case 12.—M. M., female, age 11. Admitted on April 2, 1925, with lacerated thigh and shock. The skin was reflected from the whole of the front of the right thigh. Compound fracture of the left femur. Deep lacerated wound of the left popliteal space. 7.30 p.m.: Gum 14 oz. 10.30 p.m.: Blood 1 pint. 12.0 midn.: Operation—gas and oxygen. Guillotine amputation of the left thigh. Suture of the left thigh wound. A.G.G.S. 10 c.c. A.T.S. 1500 units. Blood 1 pint.

April 4, 5, 6.—Extremely ill. Pulse 120, temperature 100°.

April 8.—Right thigh wound laid open. A.G.G.S. 30 c.c.

April 10.—A.G.G.S. 30 c.c. intravenously. Blood 1½ pints.

April 11.—Cultures gave mixed growth of *B. coli communis*, *B. welchii*, and *Str. longus*.

April 12.—Pulse 120, temperature 102°.

April 29.—Blood 12 oz.

May 8.—Skin-graft.

June 6.—Skin-graft.

July 4.—Skin-graft.

Aug. 17.—Skin-graft.

Oct. 5.—Discharged.

From the description of these cases an idea can be obtained of the symptoms and signs and clinical course of a straightforward case of gas gangrene. Such cases were, of course, well known in the War, and have been very fully described in the Report of the Medical Research Committee, 1919,¹ but it would be well to emphasize some of the more important points here and now.

Symptoms and Signs.—The disease occurs a variable time after the reception of a wound, particularly if this is lacerated and associated with much trauma and hæmorrhage into the tissues, or with fracture of a bone. Loss of blood leading to shock, and any previous disease tending to impair the blood-supply to the part (e.g., diabetes, or arterial disease), are predisposing factors. It is most common after street accidents, but may occur in any wound contaminated with dirt or fecal material.

One of the earliest symptoms is pain, coming on about thirty-six to forty-eight hours after the injury, and becoming progressively more severe. A marked rapid rise of pulse and temperature about the same time are to be expected. Such a combination of symptoms should lead to a very careful inspection of the injured part, when any of the following signs may be elicited: crepitation extending centrifugally from the wound, great tenseness of the skin, with alternating anæmic and discoloured reddish-black patches,

exquisite tenderness, a tympanitic note, and a brownish-yellow evil-smelling discharge from the wound.

If the diagnosis is not established at this stage, and the condition is allowed to progress, all the signs of profound collapse are noted, especially a great fall of blood-pressure, a greyish complexion, coldness of the extremities, cold sweating, and yet withal no loss of consciousness. The temperature falls at the end. (The picture at this stage resembles closely that presented in a case of peritonitis or late intestinal obstruction, as has been pointed out by Williams.²)

These features are stressed because it is only by familiarity with them that gas infections are likely to be recognized in non-accident cases, and it is recognition of the early symptoms—notably, the striking rise of pulse and temperature, and the severe local pain—that will bring cases under treatment sufficiently early to hold out any hope of success.

NON-ACCIDENT CASES.

The 11 non-accident cases were as follows:—

	CASES
Appendicectomy	13, 17
Appendicectomy and herniotomy ..	14, 20
Operation for ventral hernia ..	15
Colostomy	16
Ileostomy	18
Amputation for senile gangrene ..	19
Plating of fractured radius ..	21
Ischiorectal suppuration ..	22, 23

Case 13.—J. H. B., male, age 27, medical student. Highly nervous temperament.

HISTORY.—During the war had the right foot crushed by a heavy weight. Had several manipulations and subsequent immobilizations. Three years on crutches.

April 19, 1927.—Admitted for chronic inflammation of appendix.

April 26.—Appendicectomy. Pulse 80, temperature 98°, respirations 20.

April 27.—Pulse rose after operation from 100 to 140 in twenty-four hours, and temperature to 102°. Patient very restless and convinced he was going to die. Vomiting. No signs in abdomen. Required much sedative.

April 28.—Pulse 130, temperature 99.8°. Much vomiting. Sodium bicarbonate in quantities given by mouth. Attempts made to get bowels opened.

April 29.—Pulse 110, temperature 98.4°. Great restlessness continued. Patient refused to keep bedclothes or dressings on. Some vomiting. Rectal saline given. Stomach washout. Abdomen somewhat distended. Bowels opened twice.

April 30.—Pulse 150, temperature 102°. Some distension of abdomen. Occasional vomiting. Bowels opened four times. Rectal salines not well retained. Subcutaneous salines exhibited.

May 1.—Pulse 130, temperature 102°. Continuous aspiration of stomach by Ryle's tube and Sprengel's pump, 20 oz. in twenty-four hours. Hiccup started. Vomit 30 oz. in twenty-four hours. Subcutaneous saline 2 pints. Fluid by mouth 40 oz. Bowels opened six times.

May 2.—Drainage 3 pints. Vomit 2 pints. Bowels opened twice.

May 3.—Pulse 110, temperature 97° (axilla). Persistent hiccup and great restlessness though vomiting stopped. Subcutaneous salines.

May 4-9.—Hiccup persisted, but vomiting ceased. Temperature subnormal (axilla), pulse 100-110. Diarrhoea. Subcutaneous saline. Fluid intake good.

May 9.—Crepitation noticed in abdominal wall. Incised freely, with exhibition

of flavine, and Carrel-Dakin tubes. Culture of blood, negative; of wound, streptococci, *B. coli communis*, and *B. welchii*.

May 10.—Death. Terminal rise of pulse to 140.

POST-MORTEM.—Peritonitis. Mixed culture of *B. welchii*, streptococci, and *B. coli communis*.

Case 14.—A. P., male, age 22. Admitted on Jan. 2, 1927, for right inguinal (oblique) hernia of three weeks' standing. Operation for acute suppurative appendicitis four and a half years before.

OPERATION (Jan. 3).—For hernia. Apart from some omental adhesions in the sac, the herniotomy was simple and straightforward and excision of the sac only was performed.

Jan. 4.—The pulse rose steadily after the operation, and within twenty-four hours had mounted from 80 to 160. The temperature rose from normal to 102°. Much pain was complained of in the wound, and local examination showed crepitations radiating for some distance around this. Diagnosis of gas gangrene made.

OPERATION.—Multiple incisions made through the subcutaneous tissue—gas being liberated upon opening up the tissue planes—and antiseptic dressings applied. A.G.G.S. 80 c.c. intravenously, with 500 c.c. sodæ. bicarb. A.G.G.S. 40 c.c. intramuscularly and subcutaneously. Immediate and rapid improvement in general and local condition was noticed, the pulse falling to 110 and the temperature to normal in twenty-four hours, but there was a fluctuating temperature (up to 101°) for two and a half weeks. This settled later to normal. Cultures from the wound remained sterile throughout.

At the end of four weeks, however, signs of acute intestinal obstruction developed.

Jan. 30.—Operation revealed bands thought to be due to the old acute appendicitis, with a curious glairy coagulum among the intestines.

Death followed three days after the operation, and further peritoneal adhesions were disclosed.

Case 15.—R. A. H., male, age 50. Admitted on May 31, 1927, with a ventral hernia, following the drainage of an appendicular abscess, with peritonitis.

June 1.—Operation for hernia. The appendix stump was inspected.

June 4.—Pulse and temperature started rising. There was much abdominal pain, some vomiting, and complete constipation. Obstruction due to a band was diagnosed, and the slight crepitation about the incision thought to be of no account. Laparotomy was performed, and one or two bands were found and divided with the fingers inside the abdomen.

June 5.—Pulse 150, temperature 101°. There was now very extreme abdominal distension, associated with a great amount of pain. Constipation persisted, and vomiting continued. Signs of collapse were manifest. A colostomy was performed under local anæsthesia. Much gas escaped, to the relief of the patient, but he died thirty-six hours later. *B. welchii* was grown from the abdominal wound.

Case 16.—A. F. B., male, age 62. Admitted on June 27, 1927, for acute intestinal obstruction due to carcinoma of the colon. A colostomy was performed—the growth being inoperable and the patient's condition extremely bad. Temporary improvement followed.

July 2.—The condition again became grave, and, in spite of the fact that emphysema was noticeable about the colostomy wound, it was thought inadvisable to administer serum, a fatal issue being imminent.

July 3.—The patient died, and peritonitis was found post-mortem. Unfortunately cultures were not taken.

Case 17.—D. F., male, age 8. Admitted on Oct. 13, 1927, with acute appendicitis. Pulse 90, temperature 101°, respirations 22.

OPERATION.—Gridiron incision. Appendicectomy. Appendix acutely inflamed but not gangrenous, containing pus in its distal half. No peritonitis.

Two days after the operation great pain was complained of in the wound. The pulse rose steadily throughout the day, from 90 to 120. Temperature 100°. On

examination the skin was found to be tense, and marked crepitation was felt in subcutaneous tissue on both sides of the wound. A gas infection of the wound was diagnosed.

Oct. 15.—Free incisions were made above and below the wound, and gas liberated from the subcutaneous tissue. The area was swabbed with flavine, and Carrel-Dakin tubes were inserted for subsequent flavine percolation. A.G.G.S. 10 c.c. was injected locally and 30 c.c. intravenously. Recovery was rapid and uneventful.

Case 18.—E. M., male age 59. Admitted on Feb. 5, 1928, for subacute intestinal obstruction of seven days' duration. Pulse, temperature, and respiration were normal. As some relief had been obtained by enemata and as there was a clinical history and X-ray evidence of aortic aneurysm, operation was deferred. The following day, however, obstruction became complete, and laparotomy revealed obstruction due to a band. This was freed and ileostomy performed.

The general condition remained poor, and the patient died rather unexpectedly twenty-four hours later, temperature and pulse being steady.

POST-MORTEM.—No peritonitis. Marked gas infection of the abdominal wall. *B. welchii* grown in culture.

Case 19.—T. C., male, age 72. Admitted on Sept. 18, 1928, for (dry) senile gangrene of the third and fourth toes. The little toe had previously been amputated for sepsis, and there was an ulcerated patch over the head of the first metatarsal. Pulse of dorsalis pedis poor.

Sept. 21.—Amputation through mid thigh. Practically no bleeding from stump. Arteries very thickened. After the operation the patient—normally weak—grew rapidly weaker, the pulse accelerating from 90 to 130 in twenty-four hours, the temperature being, however, subnormal. Died thirty-six hours after operation.

POST-MORTEM.—Large bullæ containing blood-stained fluid were present over the stump, and gas was found in the soft tissues.

Case 20.—W. J., male, age 38. Admitted on May 24, 1928, for right inguinal hernia.

OPERATION.—Herniotomy with appendicectomy through hernial incision.

Two or three days later there was marked pain in the wound, with increased pulse-rate and temperature. The skin was tense and very tender, and crepitation was marked.

OPERATION.—Free incisions were made into the subcutaneous tissues, and gas escaped. Flavine dressings were applied and Carrel-Dakin tubes inserted for subsequent irrigation. Serum, 30 c.c., was administered locally (subcutaneously). Pulse and temperature rapidly fell to normal, and the patient made an uninterrupted recovery.

Case 21.—F. A., male, age 34. Admitted on June 23, 1928, for a simple fracture of the right radius and ulna in the middle of the shaft, with some displacement. The arm was manipulated under anaesthesia, and a plaster splint applied in supination. The patient was discharged, and kept under observation.

Aug. 27.—Re-admitted owing to the absence of evidence of union, with angulation at the site of fracture. Open operation was decided upon.

Sept. 3.—Open operation performed. The bones were brought into satisfactory position, and a plate was applied to the radius.

Sept. 4.—Within thirty hours of the operation the temperature had risen to 105°, and the pulse from 70 to 150. Great pain was complained of in the arm, and collapse was marked. Inspection showed much swelling of the arm, great tension, and a patchy mottled discoloration of the skin. Crepitation was freely elicited all over the forearm, and percussion gave a tympanitic note. The local and general signs had become rapidly worse within a few hours.

Sept. 4.—10.0 p.m.: Operation. It was thought that, in spite of the acuteness and severity of the infection, every effort should be made to save the right arm of a young man. Multiple incisions were therefore made into the limb, and much gas

and evil-smelling blood-stained fluid escaped. Hydrogen peroxide and flavine were freely used, and Carrel-Dakin tubes inserted. A.G.G.S. was given, 10 c.c., into the arm, and 40 c.c. intravenously.

Sept. 5.—A.G.G.S. 20 c.c. intramuscularly.

Sept. 9.—A.G.G.S. 10 c.c. intramuscularly.

The patient rapidly improved, and left hospital on Oct. 10.

Case 22.—R. M., male, age 22. Admitted on July 29, 1929, with right ischio-rectal abscess and extensive cellulitis of the buttocks and back. The abscess had burst seven days previously, and now foul-smelling discharge was practically continuous. Heat, redness, swelling, tenderness, and subcutaneous emphysema of the right buttock extending up to the third lumbar spine and continued on to front of abdomen. Temperature 102°, pulse 108, respirations 40. Toxæmia very marked. Slight neck rigidity and positive Kernig's sign.

July 30.—Ischio-rectal sinus laid open. Multiple incisions into buttock and back. A.G.G.S. 40 c.c.

July 31.—Emphysema spreading down right thigh as far as knee and up to sixth thoracic spine. A.G.G.S. 25 c.c. A.T.S. 20 c.c.

Aug. 1.—Further incisions. A.G.G.S. 25 c.c.

Aug. 2.—Improvement. A.G.G.S. 25 c.c. daily until Aug. 8.

Aug. 6.—Emphysema spreading down leg. Further incisions.

Aug. 8.—Pulse 100. Temperature normal, 98°–100°, for first time. Patient brighter. Cultures gave a growth of *B. coli communis*, *Str. longus*, and *B. welchii*. Improvement slow and steady.

Sept. 24.—Discharged. All wounds healed.

Case 23.—J. G. B., male, age 48. Admitted on Jan. 1, 1924. The history was vague, but it appeared that a swelling of the scrotum had been noticed eight days before (while the man was at sea). A discharging wound in the ischio-rectal fossa had been present for most of this time.

On admission the patient was in a state of profound collapse, with a small thin pulse (130) and a subnormal temperature (95°). He was, in fact, moribund. There was a wound in the right ischio-rectal fossa exuding an extremely foul-smelling discharge. Extending from this there was great swelling of the scrotum and the back of the right thigh. The scrotum was a bluish-black colour, and the thigh a dusky red. Great tenderness was present.

Operation.—Shortly after admission. Multiple incisions were made, and much skin excised. Gas escaped from all the wounds. Cultures grew *B. coli*. The patient failed to rally and succumbed rapidly.

Post-mortem.—Gas was found in the walls of the ileum.

The first case in this series (*Case 13*) was the one that drew our attention to the possibility of a gas infection following a straightforward operation. Here a healthy medical student was operated upon for a subacute inflammation of the appendix. The operation was carried out with the greatest care, and an appendix with only the slightest traces of inflammation was removed. The after-history of this case has been related in detail above, but the salient points were intractable vomiting, yet with no suggestion of intestinal obstruction; moderate distension; but, strangely, no abdominal pain. The patient died, in spite of heroic treatment, and diffuse *B. welchii* peritonitis was found at autopsy.

Cases 14, 17, and 20 demonstrate the occurrence of superficial gas infection following an appendicectomy. These all responded to free incisions and the administration of serum, though *Case 14* died subsequently of 'intestinal obstruction'. It is not unlikely that this was in reality due to a *B. welchii* peritonitis, for there are no records of cultures taken post mortem.

Case 15 was characterized by extreme abdominal distension. It is noteworthy that the appendix stump was inspected at the time of the operation for hernia. *Case 16* followed a colostomy, and *Case 18* an ileostomy, whilst *Cases 22* and *23* followed ischiorectal suppuration. These are not difficult to understand in view of the well-recognized presence of *B. welchii* in the lower bowel. In *Case 19*, however, the infection followed an amputation for senile gangrene (dry), and in *Case 21* an open operation on a bone—performed with the greatest attention to the ‘no-touch’ technique.

Bacteriology of Gas Gangrene.—Many organisms have been found in this infection, and the summary of experiences during the Great War points to *B. welchii*, *vibrio septique*, and *B. oedematis* being most frequently present. It may be suggested that in the above series these organisms were not found in every case, but it must here be pointed out that in many instances the urgent operations were performed at night or at other times when the bacteriological staff was not available, and this apparent discrepancy may therefore be explained by the fact that the suitable cultural methods were not immediately made use of. This would inhibit the growth of these anaerobic organisms and so militate against a subsequent successful culture. It is also recognized by the writer that emphysema is occasionally found around a wound that is otherwise satisfactory, but it is suggested that the cases presented are genuine cases of gas infection—some proved beyond doubt, and the others showing the clinical picture of emphysema, discoloration, marked pain, and constitutional disturbances.

Pathology.—The spread of infection is usually by direct continuity, especially along muscle fibres, with a disinclination to penetrate fascial coverings. It has, however, been pointed out by Kettle³ that dissemination of organisms may take place by: (1) Direct extension in loose areolar and connective tissue; (2) Growth along lymphatic vessels; (3) Invasion of the blood-stream. This is significant, for otherwise it would be difficult to explain certain cases in this series, particularly those following operations not in any way associated with the gut. (Four instances have been elsewhere described of *B. welchii* cholecystitis when the organisms have presumably spread to the gall-bladder via the bile-ducts.⁴)

It is not necessary to enter here into a detailed description of the histology of the lesions nor into a discussion upon the chemical changes leading up to the evolution of gas, for these have been dealt with respectively most fully by Kettle³ and Henry.⁵

Treatment.—The most radical treatment of gas gangrene is by excision of the affected part. This is easily carried out in the limbs, and is obviously indicated in cases of severe peripheral infections, care being taken to excise all affected muscle tissue. In those instances, however, where the infection is established on the trunk, complete excision is not possible. Good results may nevertheless be obtained by early incisions, irrigation with hydrogen peroxide and flavine, and the administration of large doses of serum.

The use of serum in the treatment of peritonitis and intestinal obstruction, as recommended by Williams,² is now generally accepted as a valuable aid in treatment. Apart from manifest cases of gas gangrene, great success

has followed its use in certain cases of unexplained extreme abdominal distension with marked constitutional disturbance following straightforward abdominal operations, e.g., cholecystectomy. A similarity must be remarked between such cases and *Cases 13, 14, and 15* of our series. It is felt that early administration of serum in these three cases might have helped towards averting a fatal issue. Prophylactic injections of serum should always be used in severe street accidents.

SUMMARY.

1. Twenty-three cases of gas gangrene are described, 12 following accidents, and 11 following ordinary operations.

2. The clinical signs and symptoms are discussed, drawing especial attention to severe local pain and marked rise of pulse and temperature, indicating the importance of early diagnosis.

3. The bacteriology and pathology are briefly considered.

4. The treatment is indicated, with particular reference to the value of early and plentiful administration of serum, especially where radical removal is impossible.

I am indebted to the surgeons of Guy's Hospital for permission to publish these cases.

REFERENCES.

- ¹ *On the Anaerobic Infections of Wounds, and the Bacteriological and Serological Problems arising therefrom*, Medical Research Committee, Special Report Series No. 39, 1929.
- ² WILLIAMS, B. W., "The Importance of Toxaemia due to Anaerobic Organisms in Intestinal Obstruction and Peritonitis", *Brit. Jour. Surg.*, 1926, xiv, 295.
- ³ KETTLE, E. H., "The Histopathology of Gas Gangrene", Appendix to Report of Medical Research Committee (quoted above).
- ⁴ PEARCE GOULD, E., and WHITNEY, L. E. H., *Brit. Jour. Surg.*, 1927, xiv, 646; *Ibid.*, xv, 158 (footnote).
- ⁵ HENRY, HENRY, "On Some Anaerobes found in Wounds and their Mode of Action in the Tissues", *Brit. Med. Jour.*, 1917, i.

THE APPROACH TO THE HIP-JOINT: A CRITICAL REVIEW AND A SUGGESTED NEW ROUTE.*

By R. P. OSBORNE,

DEMONSTRATOR OF ANATOMY IN THE UNIVERSITY OF MANCHESTER.

THE relatively small size of the hip-joint, no less than its great depth from the surface and its relation to very important structures, all conspire to render surgical approach a matter of some difficulty. This difficulty is well illustrated by the great number of routes which have from time to time been described by different operators. Moreover, it is evident that no one route will fulfil all conditions—a route which is ideal for drainage of the joint may prove quite unsuitable for plastic work or arthrodesis.

The following account represents an attempt to evaluate the variously named routes which have been described. This has been done by practising the operation on the cadaver in strict accordance with the respective authors' original descriptions. Each operation has been performed at least three times, and in the case of the more important routes has been done oftener than this. As the outcome of this experience it is possible to express an opinion as to the advantages, the difficulties, and more particularly as to the access given by these various approaches. A survey of the literature shows that the routes may be grouped as follows: (1) *Anterior*: Barker, Lücke, Hoffa-Lorenz, Albee, Nélaton, Hüter. (2) *Antero-lateral*: Smith Peterson, Larghi. (3) *Lateral (Trochanteric)*: Murphy, Robert Jones, Ollier. (4) *Posterior*: Kocher, Langenbeck.

1. *Anterior Group*.—All the approaches by this route conform to a common type. They approach the joint from the front by vertical incisions downwards from the anterior superior iliac spine. The well-known route of Barker enters between the tensor fasciæ femoris and the sartorius, and reaches the joint between the glutei and rectus femoris. Nélaton's route is the same, but he advises retraction of the psoas inwards, a manœuvre which is difficult and does nothing to improve access.

The approach of Lücke depends on outward retraction of the sartorius and rectus femoris, with inward retraction of the psoas. The Albee route enters between the sartorius and rectus femoris, whilst in the Hoffa-Lorenz method the incision is placed more laterally and the joint is approached between the glutei laterally and the tensor medially. The route known as Hüter is identical with that of Barker.

Of these operations by the anterior route it may be said that they are all easy, bloodless, and inflict a minimum of damage, but the access gained is very limited and full exploration is much hampered. They will suffice

* From the Department of Anatomy, University of Manchester.

for the evacuation of pus, but do not give good drainage, and are certainly insufficient for the thorough opening up of a lesion such as acute epiphysitis.

2. Antero-lateral Group.—Smith Peterson and Larghi: the essential feature of both these approaches is that the glutei are detached close to their origins from the ilium and are thrown downwards, backwards, and outwards in one mass. The exposure of the joint is excellent, and although the wound is very extensive, hæmorrhage is easily controlled. From experience of this operation in the living, it would seem to be the route of election for all plastic, arthrodesis or 'shelf' operations. It is to be remembered that it is at the best lengthy, and that shock is by no means negligible.

3. Lateral Group.—The methods of Murphy, Robert Jones, and Ollier, subject to differences in skin incisions, are essentially the same in principle: they depend upon an osteoplastic resection of the great trochanter. After division near its base the trochanter with its attached muscles is reflected upwards. The operations are easy of performance, the exposure is good, any part of the acetabulum or upper end of the femur is easily reached, and provided the trochanter be not divided too low down, the loss of blood is small. The re-fixation of the trochanter is a complication, and may indeed be difficult, needing tenotomy, if the position of the hip has been of necessity altered by the operation. These are obviously unsuitable procedures in infected cases.

4. Posterior Group.—This includes the operations of Koehler and Langenbeck. In the route devised by Koehler an incision is made from the posterior margin of the base of the trochanter major upwards to the postero-superior angle of the trochanter, and thence to the posterior superior iliac spine. The gluteus maximus is then divided in the line of its fibres and the edges are retracted. The gluteus medius is separated at its insertion into the trochanter major and thrown upwards. The pyriformis, obturator internus, and gemelli are divided at their insertion into the trochanter and thrown inwards. The capsule is incised, and then the superior half of the trochanter major is divided with a saw from the main bone and thrown upwards with its attached muscles. By adducting the diseased limb across the sound one and rotating outwards, the head of the femur is dislocated posteriorly.

Anatomically this is an excellent approach; the exposure of the head and neck of the femur and the joint cavity is exceptionally good. The operation is not difficult to perform and drainage is good, but section of the trochanter renders the procedure unsuitable for infected cases.

In Langenbeck's approach an incision is made from the posterior superior iliac spine to the postero-superior angle of the great trochanter. The gluteus maximus is divided in the line of its fibres and the edges are retracted. This exposes the posterior margin of the gluteus medius and the superior margin of the pyriformis. These are retracted, and if necessary the pyriformis is divided or loosened at its insertion. This is a rapid and easy approach with little disturbance to the tissues. The area of joint exposed is, however, limited, as the superior margin of the tendon of the pyriformis is at least one-eighth of an inch above the postero-superior margin of the neck of the femur. This factor also makes drainage difficult.

Dixon recommends for further exposure the loosening of the gluteus medius and minimus at their insertions, and then retracting them backwards. The division of all these muscles will, however, weaken the stability of the joint.

SUGGESTED NEW OPERATION.

As the outcome of many operations on the cadaver, I venture to suggest the following route. In view of the fact that Langenbeck's method is less mutilating than Koehler's, but the exposure somewhat limited, I have sought to make a combination of the two operations. The method adopted depends on the observation that the tendon of the pyriformis runs along the postero-superior margin of the neck of the femur, and the gemellie mass runs obliquely across the neck.

Incision.—With the diseased limb adducted across the sound limb, incise from a point $1\frac{3}{4}$ in. below the posterior superior iliac spine to the postero-superior angle of the great trochanter and then down the posterior edge of the trochanter for two inches.

Steps in the Operation.—The subsequent stages of the operation are :
(1) Divide the gluteus maximus in the line of its fibres and retract the edges.

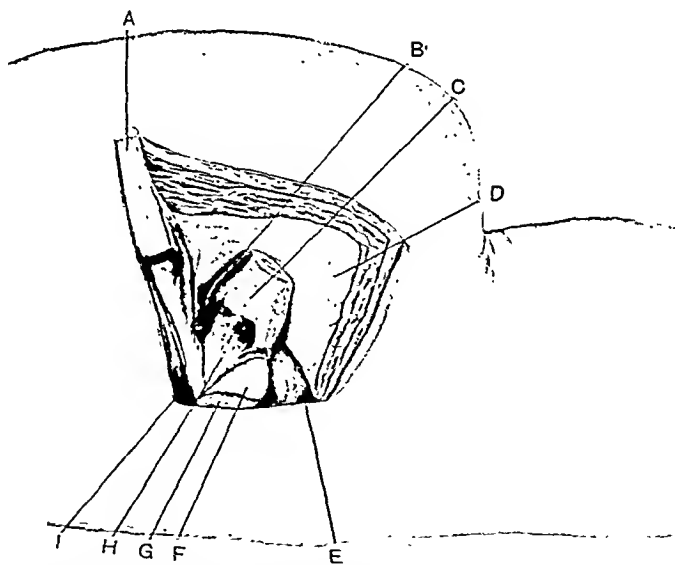


FIG. 32.—Illustrating the approach at stage 4. A, Tendon of pyriformis; B, Branch of sciatic artery; C, Gemellie mass; D, Reflected gluteus maximus; E, Quadratus femoris; F, Neck of femur; G, Great trochanter; H, Capsule of hip-joint; I, Gluteus maximus.

(2) It will be found that the first part of the incision corresponds to a line just above the lower edge of the pyriformis. (3) Internally rotate the limb and divide the tendons of the pyriformis and the gemellie mass close to their insertion, and throw the two inwards to be held by an assistant. (4) The capsule of the joint is now exposed and may be incised (Fig. 32). (5) Further

exposure can be gained, if required, by retracting the quadratus femoris downwards and the gluteus medius upwards.

There is no difficulty with hæmorrhage, but the following vessels will be encountered: (1) A branch from the sciatic artery which runs between the tendon of the pyriformis and the superior gemellus (*see Fig. 32*). (2) The ascending branch of the internal circumflex artery. (3) A branch of the gluteal artery running along the upper border of the pyriformis; this should be retracted upwards. It will be noted that the reflection of the gemellie mass protects the sciatic nerve mass from injury.

The advantages of this route would appear to be: (1) Almost the whole of the posterior surface of the joint and the neck of the femur is exposed. (2) There is no interference with the great trochanter (*cf. Kocher*). (3) The displacement of the tissues is slight and easily reparable. (4) There is adequate provision for drainage.

As the result of this investigation I would suggest that for all 'quiet' work the Smith Peterson is the route of election, but that for 'infected' cases of any type requiring a direct attack on the joint or head and neck of the femur with subsequent drainage, a posterior route such as I have suggested will be found advantageous.

IS IT TRUE THAT THE VALVES IN A VEIN NECESSARILY BECOME INCOMPETENT WHEN THE VEIN DILATES?

By R. E. KELLY,

SENIOR SURGEON AT THE ROYAL INFIRMARY, LIVERPOOL.

IN discussions on the well-known incompetency of the valves in varicose veins the statement is often made that the valves become incompetent in consequence of the dilatation of the veins.

The subjoined figure (*Fig. 33, A, B*) shows that in dilatation of the veins due to another cause the valves remain perfectly competent, though the diameter of the vein has increased at least fourfold. The photographs are taken from a man with a well-marked arteriovenous aneurysm of the radial artery in the anatomical snuff-box. In *Fig. 33, A* shows the very marked dilatation of the superficial radial vein, while in *B* the lower end of the vein has been compressed and the vein emptied by stripping it in an upward direction.

The vein remains empty. The valves in it are therefore competent. It is a repetition of Harvey's observation, but on a markedly dilated vein, and proves that dilatation of a vein does not necessarily produce incompetency of its valves. I have made this observation on many cases of arteriovenous aneurysm, but never before demonstrated it so graphically.

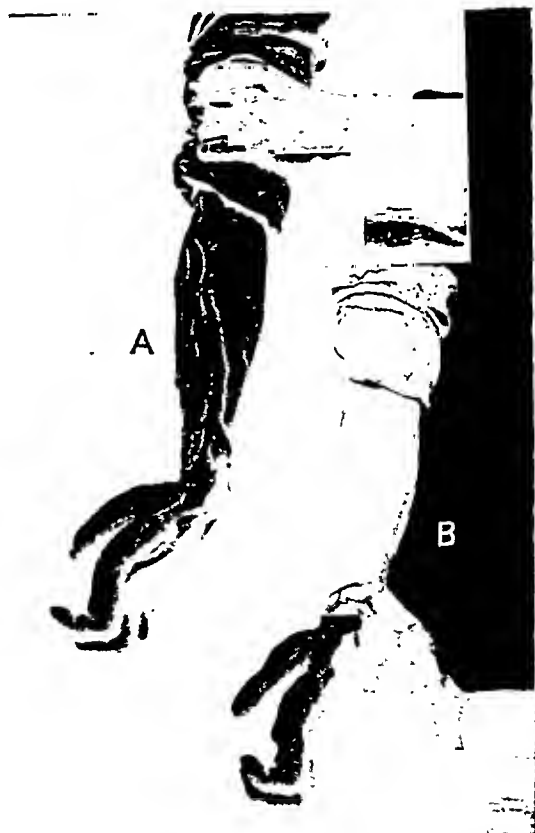


FIG. 33.—A, Dilatation of radial vein : B, Vein emptied by stripping, showing competency of valves.

THE SPLINTING OF CASES OF TUBERCULOSIS OF THE HIP.

By M. FORRESTER-BROWN,

VISITING SURGEON TO THE CHILDREN'S ORTHOPÆDIC HOSPITAL, BATH.

THE tour of a series of hospitals, whether general or orthopædic, where cases of tuberculosis of the hip are under treatment, shows that there is as yet no standard method of treatment common to all, or even a majority, and this may be presumed to mean that the most efficient method still remains to be discovered. In most spheres, it is the subject which is still debatable that is the most interesting, and therefore this article is written, not in the hope of laying down the perfect method, but in order to invite further thought and discussion over a subject of extreme complexity and importance.

The discussion in the Orthopædic Section of the British Medical Association at the Annual General Meeting in Manchester in 1929 on the operative treatment of tuberculosis of the larger joints, served to focus attention again on this question of the hip-joint; but the limited time available and the deliberate restriction of the debate did not allow the full subject even to be sketched out. It did, however, reveal the interesting, if depressing, fact that the open-air hospital by itself has not solved the problem of the treatment of tuberculosis of the hip, as some of its more optimistic supporters had originally hoped it would. This is not to say that open-air and sunlight treatment are not valuable adjuncts, indeed essential features of the treatment, but that they must be supplemented by skilful splinting and in many cases by operative methods. This, which is now common experience, was pointed out many years ago by one of the pioneers of sunlight treatment, Bernhardt, of St. Moritz; but it is doubtful whether the fact is recognized by the rank and file of the profession or by the general public.

The variety of methods of splinting in use for tuberculosis of the hip can be partly explained by the fact that this disease presents a number of diverse types, and also in its prolonged course goes through a series of different stages, each demanding different handling. As these types and stages are not separated by hard-and-fast lines of demarcation, it is easy to understand that a method which is in fashion in one institution and which gives a large proportion of successes is applied indiscriminately to all. It seems likely, however, that our results could be improved by attention to this aspect of the question.

Statistics are tricky things to handle, and, with so slow a disease, it would take a lifetime to collect the material on which to base conclusions at all sound or safe. On the other hand, the careful study of even a few typical cases may throw enormous light on a disease—a fact which has been demonstrated by all the pioneers of medicine and surgery. Accordingly, the writer

has appended to this article the summaries of a series of case records which illustrate the points under discussion, i.e. : (1) The difficulty of diagnosing with certainty whether a hip is tuberculous, both in the very early and the very late stages ; (2) The necessity of including operative interference in our schedule of treatment even with tuberculosis of the hip ; (3) The advantages and drawbacks of various methods of splinting, including a new ambulatory splint, designed by the author and published here for the first time.

DIFFICULTIES IN DIAGNOSIS OF TUBERCULOSIS OF THE HIP.

As all rational treatment must be based on correct diagnosis, the first essential before deciding on any type of splinting is to settle, if possible, whether the case is tuberculous or not. In this matter, unfortunately, the greater our experience the less our confidence in our power to decide. The difficulty of the clinical diagnosis is so great that a number of authorities say that biopsy should be resorted to early and in all cases of doubt ; but unluckily even this only settles the point in a certain number of cases ; because in some instances the piece of tissue removed may come from a part of the joint as yet uninvaded by the tubercle bacilli, or their numbers may be so few as to evade observation. Though it is sometimes wise to resort to this method, its use does not really solve the whole problem.

There seem only two rational alternatives : (1) To treat the case as non-tuberculous until definite evidence appears that it is ; (2) To treat the case from the moment of doubt as tuberculous, and then cautiously test the amount of freedom from restraint that it will tolerate without relapse. The former method is risky, because the first certain signs of the tuberculous nature of the case may be destruction of bone, which is irreparable. The latter method has the disadvantage of involving expensive in-patient treatment for months for a number of cases which eventually prove to be non-tuberculous and so probably susceptible of cure by cheaper means. This is a dilemma which faces us with almost every disease—for instance, quite a number of cases of appendicitis would recover without operation, but if more were left to conservative methods, how many would perforate and have a stormy convalescence or fatal issue ? It is all a matter of refinement of diagnosis. The rapid organization of medical treatment which is occurring at present is adding to our immediate difficulties by providing us with numbers of cases in stages so early as to be unfamiliar to us ; but no doubt in due course this will lead to greater refinement of diagnosis.

Early Stages.—Tuberculosis of the hip in its earliest stage is either a *synovitis*, which is indistinguishable from any other synovitis, or is a focus *confined to the bone*, which may irritate the neighbouring synovia and so lead to vague joint symptoms, but is diagnosable only by X rays. *Case 4* was of this nature. *Case 1* was a synovitis suggesting tuberculosis, but ultimately proved to have another origin. *Case 3* probably began as a tuberculous synovitis, but later there was invasion and destruction of the upper part of the acetabulum. *Case 2* was suggestive for a long time of synovial tuberculosis : but while the clinical signs were still vague, X rays showed an advanced stage of coxa plana, a disease in which the epiphysis itself is attacked by a

degenerative process of unknown origin, and the joint is involved through the subsequent deformity, but not, as far as is known, in the first instance, for X rays show a clear joint-interval all through, although both the head of the femur and the acetabulum change their shape. In synovial tuberculosis, even in the early stages, the joint-interval becomes narrowed (though there may be a brief period of widening, when effusion is present) and the capsule is marked out by a shadow of abnormal density.

Final Stages.—Difficulties in diagnosis may also occur in the final stages of tuberculosis of the hip, when there has been considerable destruction of the head of the femur, probably with subluxation upward, or even complete dislocation. The same X-ray appearances may result from an old suppurative arthritis, or osteomyelitis invading the joint, though these two conditions usually show a degree of new bone formation which is not found in tuberculosis; while the dislocation which results from them tends to leave a lax joint, whereas the dislocation in tuberculosis has been produced by a slow process accompanied by the formation of much fibrous tissue, which makes the false joint stiff. The importance of the diagnosis from the point of treatment is that, if operative correction is considered, there is the risk of lighting up the original infection. If this was originally acute, there is for many months a serious risk of a dangerous flare-up even after a simple operation such as osteotomy; but after several years it is probable that even an extensive arthroplasty will be safe. With tuberculosis, on the other hand, an extensive opening-up of the joint at any period, however remote from the date of onset, runs the risk of freeing bacilli which are still active, although they have lurked harmlessly for years in the fibrous tissue, so that the simplest possible operative method should be chosen. As the deformity at such a stage does not yield satisfactorily to splinting alone, its full treatment cannot be discussed within the scope of this article.

CLINICAL TYPES OF TUBERCULOSIS OF THE HIP.

As already mentioned, different methods of splinting are applicable to different types of the disease, so these types must be briefly considered. They will be differentiated on clinical grounds, for they do not represent clearly defined pathological entities.

1. Primary focus in the neck of the femur (*bony*) with only slight or localized invasion of the synovia. This type has been already referred to as being represented by *Case 4*. When it comes under treatment in its early stage, it is probable that only very moderate fixation of the joint is required, and that healing depends almost entirely on the application of general measures, such as open air, sunlight, suitable diet, etc.

2. Rapid general invasion of the *synovia*. As pointed out already, these cases in the early stages are undiagnosable from any severe synovitis, and require good fixation in the joint. It is only by finding for what a prolonged period fixation is required to prevent relapse, or else by discovering the tubercle bacilli themselves through aspiration or biopsy, that the diagnosis can be made. These are the cases which pay for early, thorough, and persistent treatment; but as far as our present knowledge goes, there can be no

certainly of restoring a useful range to such a joint, even when treatment is begun early. *Case 10* probably began as such a case, judging from the X-ray appearances and history. Treatment was begun early and was thorough, and yet at the end of a year the joint was full of thin tuberculous pus and there was no fibrous barrier whatever. Splinting by a bone-graft altered the clinical and X-ray picture rapidly in a most favourable manner. Though others of the cases recorded may have begun as a synovitis with secondary invasion of the bone, at the stage at which they came under observation there was considerable destruction of bone, so that it was impossible to say where the disease started.

3. Insidious spread of the disease in the *upper part of the acetabulum*, with synovial involvement and steady subluxation upward of the head of the femur, which itself becomes destroyed later. The difficulties of treatment in this type and the poor prognosis, with tendency to relapse for years, were stressed by Perkins in his monograph on tuberculous of the hip. It is this type of case in which the tendency to subluxation has been so well prevented by Pugh at Carshalton by his special method of traction, to which reference will be made later. These cases are common, and at the stage at which they reach us it is usually impossible to say whether the primary focus was in the upper margin of the acetabulum or in the synovia. *Case 3* is an instance of this type, whose X-ray seven months after the onset of symptoms gave no definite evidence of joint or bone disease, and whose clinical signs were equally vague until definite bone destruction and subluxation had occurred. *Case 7* is a similar case which healed spontaneously with ankylosis after bursting of an enormous abscess. *Case 8* is a further example, which showed very little healing three years after onset, in spite of bursting of an abscess, but which healed and ankylosed under a year after a Hibbs's fusion operation. *Case 9* was a similar case which failed to heal with five and a half years of conservative treatment, but improved rapidly after the fusion operation, though it is too early to speak of the final result. *Case 11* was a similar one, which six years after the onset, and after almost continuous orthopaedic treatment in various institutions, showed a joint full of thin pus, and with very little evidence of local healing. This patient appeared to be in excellent general health, but failed to stand a fusion operation, and succumbed, apparently from toxæmia, about five hours after the end of the operation, developing respiratory failure, which only resulted later in cardiac failure.

STAGES OF THE DISEASE.

Having considered briefly the clinical types of tuberculosis of the hip, it is necessary to review the stages of the disease in their relation to treatment. They may be roughly divided into three, as with most other diseases: (1) *Stage of active invasion* and very little tissue resistance. (2) *Stage of local resistance*, which in this instance is chiefly by fibrous tissue formation, which forms a barrier round the infected area, diminishing its blood-supply and also imprisoning the invading organisms, but leaving them potentially active, should they be released by accident or operation at some later date. (3) *Stage of true healing*, with or without restoration of damaged tissues, and

with subsidence of all the general symptoms, such as pyrexia and loss of body-weight.

1. Stage of Active Invasion.—It is well known that this stage may last for years in the neglected or the inadequately treated case; but what seems less widely known is that it may also last for years in spite of sunlight and open-air treatment, supplemented by methods of splinting, which have been given credit for the healing of hundreds of other cases. *Cases 9, 10, and 11*, already referred to, are instances of this. *Case 7* remained in this stage for two years, in spite of treatment which had in no way been altered, when he suddenly started to improve and eventually healed spontaneously.

2. Stage of Local Resistance.—This stage may still show symptoms of general toxæmia, and in such cases its treatment demands the same general measures at *Stage 1*. It is also apt to be characterized by steady increase of deformity, for the attitude which was induced in *Stage 1* by muscle spasm is likely to be maintained by the fibrous tissue which forms, and to be aggravated by the contraction of scar tissue. One of the most difficult points to decide in treating a case at this stage is whether an increase of deformity during ambulatory treatment is due to such shrinking of scar tissue or to renewed muscle spasm representing a lighting-up of the infection. It is the writer's view that, even in the later part of this second stage, these two risks can only be guarded against adequately by splinting the hip so as to cut out any movement of more than a few degrees in any direction at all. To meet this problem in as simple a manner as possible during ambulatory treatment, the writer has designed the splint which is described in detail below.

It is in *Stage 2* that most cases have come for treatment in the past, and as deformity is fairly easy to correct in this stage, it is obvious why a whole variety of methods have been given the credit for curing the disease.

3. Stage of True Health.—This stage is not attained as often as the optimists would have us believe, when merely conservative methods are used. On the other hand, the pessimists, who say that no tuberculous joint ever recovers its full range, or attains the equally safe stage of bony ankylosis apart from operation or secondary infection, find their statements disproved by such records as that of *Case 6*, in which the diagnosis was confirmed bacteriologically and in which, after partial destruction of the head of the femur, the full range returned to the joint, and X rays suggest that the bone has re-formed. It would have been most unfortunate if an ankylosing operation had been done early to such a case, which demonstrates the need for a reasonable trial of conservative measures.

Having regard to the above considerations as to the type and stage of the disease and a reasonable degree of certainty in the diagnosis, the writer suggests the following as a safe system of treatment.

TREATMENT OF TUBERCULOSIS OF THE HIP.

1. In the active stage complete recumbency in the open air, with the minimum fixation of the hip which will induce the local and general signs of activity to subside, and with great attention to general measures, such as sunlight (natural or artificial), suitable diet, careful grading of the periods

of rest and activity (educational, social, etc.). Such treatment will need to be persisted in for about a year in most cases, much longer in some.

2. In the quiescent stage, when clinical and X-ray signs suggest steady progress of healing, gradual increase of activity, at first associated with recumbency and then for many months still without weight-bearing. When weight-bearing is first begun, the author's splint described below enables it to be tried without permitting any leverage on the joint.

If the second stage of fibrosis and healing is unduly prolonged, then one has to make the difficult decision between carrying on conservative measures for an indefinite period of years, or resorting to operative treatment with a view to producing bony ankylosis as rapidly as possible, for local conditions in the hip-joint make a true radical removal of the disease impossible.

3. The stage of true healing, whether with restoration of full movement or with spontaneous bony ankylosis, naturally requires no active treatment; but it demands very careful and regular supervision to be sure that healing is final, and that one is not merely witnessing one of the stages of deceptive quiescence that are so common in this disease.

TREATMENT OF THE ACTIVE STAGE.

Splinting in the active stage requires to provide sufficient fixation to check local irritation and its resultant muscle spasm, while it should limit as little as possible neighbouring joints, or even the hip itself, and should interfere as little as possible with the circulation in the limb and with the access of light and air to the skin.

Pugh's Method of Traction.—In the writer's experience the ideals mentioned above are best attained by Pugh's method of traction from the thigh only of the affected side, with attachment to a bar at the foot of the bed, which must be extremely tilted—i.e., a fixed traction, in contrast to that of the popular weight-and-pulley method. This method has been described in the Metropolitan Asylum Board's *Annual Report*, 1926-7, and like all others it does not give such uniformly good results in the hands of imitators as in those of its originator. Though it sounds extremely simple, there are a number of details attention to which is essential for success.

1. The adhesive strapping must be very strong, because the object of the method is to allow the child to wriggle constantly, like a fish on a hook; this keeps up the circulation in the muscles and their tone, and keeps the hip-joint mobile in all the cases where this is possible; some have so much cartilage destruction before they come under treatment that it is impossible.

Pugh himself uses a special 'moleskin' strapping, but this is complicated by the fact that different types must be used in summer and winter. In practice the writer found that if sunlight was allowed access to the limb, blisters were apt to form under the strapping; while even the winter type seemed to come off very easily in cold weather. The Sisters in the Bath Orthopædic Hospital found that the old-fashioned soap and canvas strapping held best to the skin and was strong enough not to tear. Whatever type is used, it is essential, in order to get even sticking to the skin, that no pull should be applied for forty-eight hours, during which time the thigh is kept constantly warm by hot bottles round it.

2. In order not to get an uneven pull on the skin of the thigh, Pugh points out that it is necessary to cut the two pieces of strapping so that they form a complete case, or mould, for the thigh, and they must be bandaged over very smoothly. They must be tapered towards the knee, where they are sewn to half-inch lamp-wick, which is a convenient material for tying, and, as pointed out by Dame Agnes Hunt, does not stretch as much as cord or tape.

3. The knee-joint is left quite free, which eliminates fear of pulling it lax, as may occur when adhesive is attached down the length of the limb and perhaps the upper part slips; also lateral and backward displacement is not apt to occur when the muscles are allowed to balance one another by constant gentle exercise. Directly one fixes the knee at all, one must splint it in all directions, as by a closely-fitting trough splint such as Rollier uses so successfully. Such a splint is very difficult to keep exactly in position while movement is allowed at the hip, and if it slips, sores are sure to be produced. This is a nursing problem, easier to solve under conditions where the nurses are permanent, like Rollier's, than with the British system of frequent changes.

In Pugh's method, should a blister occur on the thigh, or during the necessary changing of the strapping every few months, traction is temporarily applied to the leg, whose skin has been kept in good condition for the purpose, and the few days required are insufficient to endanger the knee-joint.

4. Rotation of the hip is controlled by putting a light shoe or boot on the foot and nailing to the heel of it a wooden cross-bar, whose ends, resting on the bed, prevent the foot from twisting, and through it the hip from rotating.

5. The sound limb is left absolutely free, so that it does not atrophy, as it does when bandaged on a frame; thus a greater area of skin is left free for sunlight treatment.

6. In order to limit the strain on the traction, it is usually best with children to apply a firm canvas restrainer across the upper part of the sternum and round the shoulders; this does not hinder respiration in any way.

7. As already noted, the traction strings, which can usefully be made of strong lamp-wick sewn to the ends of the canvas somewhere near the level of the knee, are fastened to a rod or hoop at the foot of the bed, which is raised much at its foot. The most convenient bed for the purpose is the one in the form of a wheeled carriage designed by Pugh and manufactured in the workshops at Carshalton; but if one cannot have this, a very simple method suggested by Pugh is to put one end of the fracture-board over the head-rail of the bed and use that as the foot; this allows the castors of the bed to function still, and prevents interference such as occurs when raising-blocks are used and someone forgets to put them back after moving the bed.

Advantages of Pugh's Method.—

1. In favourable cases the movements of the hip-joint are maintained throughout the treatment.

2. There is a minimum wasting of muscles.

3. A maximum area of skin is available for light treatment.

4. The patients feel themselves free and are more contented in mind than with other methods.

5. The joint area is completely open for frequent inspection.

Drawbacks to Pugh's Method.—

1. It is not always possible to keep the adhesive fixed to the thigh for reasonably long periods, and immediately it has slipped, serious damage may occur to the joint; there is, as it were, no second line of defence. This is largely a nursing problem, and doubtless with prolonged training of the staff this disadvantage diminishes.

2. Although movement may be retained and subluxation be prevented, yet spreading of the disease may occur, as evidenced by one patient in the Bath Hospital (not recorded in the series) in whom an abscess appeared very suddenly and burst after six months of the treatment, when in all other respects she seemed to be making admirable progress.

3. A certain number of cases do not keep the hip mobility, or lose an initial flexion deformity, or settle down as regards general signs like pyrexia, whereas when more rigorous fixation is applied by means of a frame they do.

Jones's Double Hip-abduction Frame.—In view of what has been said above, if, in the acute stage, Pugh's method does not result in steady improvement within the first few weeks, it seems best to nurse the case on a Jones's hip-abduction frame with fixed traction to both lower limbs, applied by means of adhesive strapping from the groins to the malleoli. When this apparatus is properly applied it eliminates all movement from the hip-joint in a way that can only be surpassed by plaster-of-Paris, and when it is handled by experts, such as sisters trained at the Shropshire Orthopædic Hospital, it is admirable. Unfortunately, very few sisters trained in other institutions seem to do justice to it. As its application is time-taking, it is impossible for a busy surgeon to give it himself the supervision it requires. The apparatus is too well known to require description here, but one or two points may be referred to briefly.

1. Unless the side-bars are fitted accurately with a wrench, lateral mobility of the trunk is not controlled, and an adduction deformity may develop insidiously.

2. Unless the knee is carefully supported by a pad, a genu recurvatum is likely to develop; while if this pad is made too thick, it will have the effect of flexing the hip; this also happens if the part of the saddle behind the joint is allowed to flatten unduly. Although bony ankylosis of the hip is most satisfactory with slight flexion, yet if flexion is allowed with fibrous ankylosis, the common ending of tuberculosis of the hip, then walking will nearly always lead to an insidious increase of the flexion till it becomes a serious deformity; so that it is wisest to aim at a straight hip, especially in children whose mobile lumbar spine easily compensates when sitting.

3. As the tractions are covered by more than one bandage, it is easy for any slipping to remain unnoticed for a day or two, which will induce serious sores on the skin and interfere with the application of fresh strapping. Although this will not happen under the care of an expert, it does happen with otherwise good orthopædic nurses.

4. Directly the groin-strap is loosened, the whole system is disconnected; yet this vital part is at the mercy of the probationer, who is constantly handling the patient. It is extraordinarily difficult to bring up the *moral*

of the nursing staff to a pitch at which they regard the groin-strap as the most sacred thing in the hospital!

So long as we keep our attention on these vital points, we shall find the abduction frame (or its modifications) one of our most valuable weapons in the treatment of a case of tuberculosis of the hip which does not settle down with partial fixation, as so many do.

TREATMENT IN THE QUIESCENT STAGE.

Treatment in the quiescent stage demands great care and judgement, for it is so easy to lose in a few weeks what has been gained by months of care. The writer suggests that the following principles are sound.

1. If a wide range of movement remains in the hip, then free movement in bed should be allowed for about three months, a restrainer being still used over the shoulders to ensure that most of the day is not spent sitting, i.e., with a flexed hip. It is good to allow several hours each day in the prone position, because the effort to see the world exercises the hip extensors and so counteracts any tendency to flexion-contraction. If this freedom produces no signs of relapse, the patient should be allowed up for short periods, but it is wise to protect the hip from accident or abuse by the splint described below.

2. If the hip is stiff, but X rays suggest that movement might still return, then either the freedom in recumbency may be tried or the patient allowed up at once in the ambulatory splint. If the parents cannot be trusted to keep the splint on and in position, it is wisest to send the patient home first in a plaster-of-Paris spica, with which they cannot tamper; then when no sign of recrudescence is found after three to six months of this modified activity, it is safe to apply the removable splint.

Plaster has the drawback of keeping light and air from the skin and inducing more muscle atrophy than a splint, which permits movement for a few degrees in any direction, sufficient to keep up some muscle tone. It is also heavy, and therefore less good for the general health of a patient who has been so long in bed. In spite of this it may be found, as in *Case 6*, that each time it is changed the range has increased.

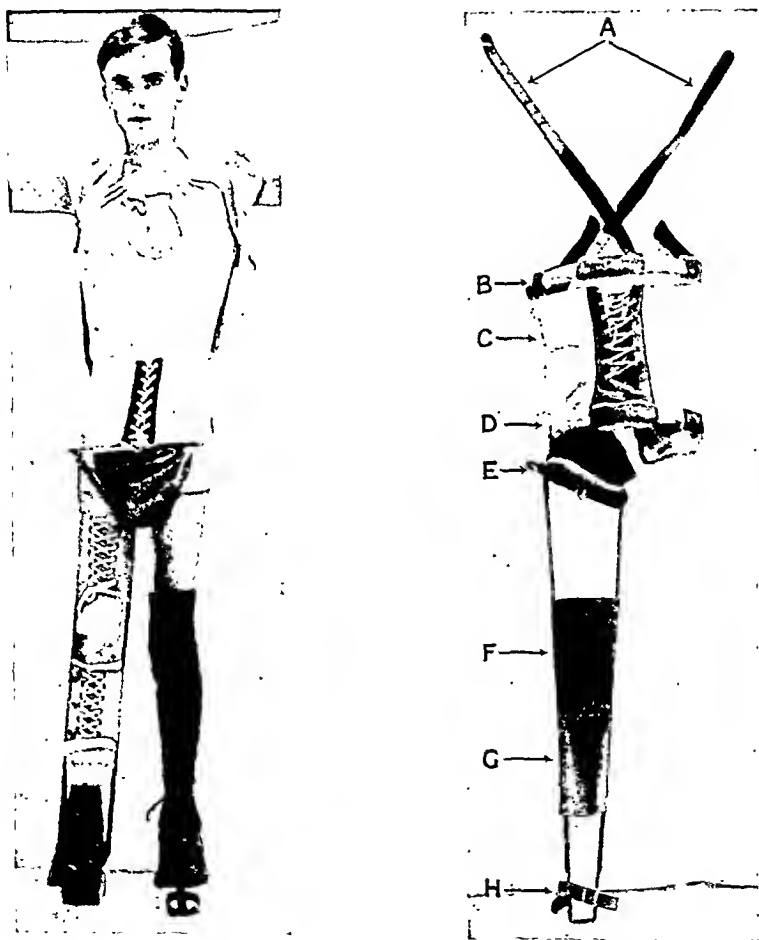
3. Even if very firm ankylosis in good position is present, it is rarely bony after spontaneous healing, though this is hard to determine, both clinically and by X rays, unless definite trabeculae are seen to pass right across the old joint line. Therefore it is unwise to allow walking unsupported at the start, and is best to allow the patient up in the author's splint, which can be discarded for increasing periods each day after the first month.

4. The really difficult case is the one which appeared quiescent after prolonged recumbency, but when allowed some liberty shows diminution of the range, or other signs of renewed activity of the disease. In such the only really satisfactory form of splinting is by a bone-graft by some technique such as Hibbs's, who moves up the great trochanter with its neighbouring bone to bridge the gap between the upper surface of the neck of the femur, which must be well rawed, and the ilium immediately above the acetabulum, which can be rawed by lifting a pedicled flap of periosteum and muscle, base upward. The writer has introduced a modification of Hibbs's original technique which seems to simplify the operation, i.e., to slide the graft upward into the bed

rawed, as above, instead of turning it upside down in order to wedge the piece of shaft under the iliac flap, as Hibbs did. Although the latter method appears to give better mechanical fixation of the graft, yet, in practice, firm suturing of the muscles over it gives all the stability required.

After the operation the patient is most conveniently nursed on Jones's abduction frame for a month or so, until healing is firm and X rays show the new callus forming. Then the patient can be allowed up, either in plaster, or in the ambulatory splint.

The Ambulatory Splint.—As numerous references have already been made to the ambulatory splint designed by the author, it can now be described—



FIGS. 34, 35.—Author's splint: front and back view (note laced canvas). A, Shoulder straps; B, Upper cross-bar; C, Abdomen corset; D, Lower cross-bar; E, Caliper ring; F, Thigh corset; G, Leg corset; H, Heel strap. (Reproduced by kind permission of the Oxford Medical Publications from the author's book *Deformities in Infancy*.)

briefly, for the illustrations will show best what it is (Figs. 34, 35). The principle is to utilize the Thomas walking caliper, with its advantage of

taking some weight off the joint, and to cut out the drawback of the caliper by attaching it to body-bars and a corset, which serve effectually to eliminate movements, beyond one or two degrees, in all directions. That they do so is shown by clinical results, a few of which are recorded in the attached case notes. It has been in constant use for over two and a half years on a large series of cases, in all of which it has succeeded in maintaining unchanged the position of the joint in which it was applied. It must be clearly understood that it is under no circumstances corrective, but must be fitted to the joint in the exact angle which has been attained by previous corrective measures. It is easiest to fit when the joint is in the neutral position, i.e., straight, neither hyperextended, nor abducted, nor adducted. The fitting is a little difficult when other positions are present, and is best attained by a tracing of the limb, assisted by a strip of lead.

The details of construction are as follows:—

The caliper has the usual hard steel ends, which can be slid up and down to adjust the length; it has the usual soft iron ring seated under the tuber ischii, and soft iron inner bar, reaching from the perineum to the heel of the boot. Both these are made of $\frac{3}{8}$ -in. mild round steel.

The outer bar of the caliper is carried right up the side of the body to the nipple line (or the level of the angle of the scapula), and, as it bears all the leverage strain in resisting flexion of the hip, is made of $\frac{3}{4}$ -in. by $\frac{1}{4}$ -in. steel, the lower end being adjusted to the steel caliper end. For heavy adults $\frac{3}{4}$ -in. by $\frac{1}{2}$ -in. steel is used.

A corresponding upright *body-bar*, made of the same steel, runs along the opposite side of the trunk from the nipple line to the top of the great trochanter.

Strong *canvas corsets* are attached back and front to the uprights to keep the lumbar spine and hip extended. As there is enormous strain on these, it was found necessary to attach them to the uprights by making the steel bars two-ply and using the canvas double, wrapping the back pieces round one piece of steel, and the front round the other, and then riveting the two together. This is a very important point, for no stitching of canvas to steels will resist the strain put upon it. Since this has been done, no corset has given way.

In order not to weaken the long upright which runs from ankle to axilla on the diseased side, it was found best to *rivet the caliper ring to the inner ply* of steel on that side, i.e., the one which carries the front corset. This has added greatly to the efficiency of the splint, for the enormous strain between caliper and body part is thus distributed through a series of rivets.

The two uprights are connected by a *top and bottom cross-bar* made of $\frac{3}{4}$ -in. by $\frac{1}{2}$ -in. mild steel, which must be carefully shaped to fit the trunk just below the angles of the scapulae and just above the coccyx; it should be padded with a furrow for the lower spines of the sacrum. To get good fitting it is best to take a tracing with a strip of lead for each of these bars, and a rough fitting should be made before the splint is finished up. Once fitted correctly, they give no trouble from pressure. Accurate fitting of the caliper ring is also essential. The thigh and leg are held in the caliper by *leather corsets*, which also must be fitted accurately.

The rotation of the hip is controlled by the angle at which the socket is

put into the *heel of the boot*. Here again no attempt must be made to induce correction, for that will only result in the production of sores. The tilt must be made to correspond to whatever degree of fixed rotation is present in the hip.

Advantages of the Ambulatory Splint.—

1. It takes a great deal of weight off the joint by means of the caliper-ring; indeed, actual traction can be obtained by lengthening the leg bars, so that the boot is partly pulled off the foot and the heel cannot reach the ground; but if this is done, very careful watch must be kept for pressure sores.

2. It eliminates all flexion and adduction movements from the hip, and discourages the patient from sitting down, though he can sit, like any case of ankylosed hip, on half of a chair. This is an advantage, for these convalescent patients are apt to spend most of the day seated, thus favouring the flexion deformity which is so apt to recur.

3. It allows attention to the skin and sunlight treatment and dressing of sinuses in a way that a plaster spica, to which it corresponds, does not.

4. It allows removal of the apparatus for part of each day and for the whole night in convalescent cases, while giving much firmer support for walking than is given by a bivalved plaster.

Disadvantages.—The chief drawback is that the splint requires very accurate fitting in the first instance. After that it wears for years. In a growing child the cross-bars may want renewal after about nine months, but the uprights and caliper seem to last at least two years. Some splints have needed no alteration till discarded after healing of the joint.

The splint was demonstrated to the British Orthopædic Association at Bath in October, 1927, when it had been in use only a few months. It was approved in principle by most of the members, and since then it has stood the test of use in all sorts of hip cases in which it was desired to eliminate movement and reduce weight-bearing, while allowing walking. It has been adopted by the writer's colleagues in the Bath Orthopædic Hospital, of whom she has to thank Mr. Mumford for permission to publish several of the cases in the series which is appended in order to illustrate in detail the points briefly referred to in this article.

CLINICAL SUMMARIES OF ELEVEN CASES.

Case 1.—N.W., female, age 10 years. Synovitis of hip (probably from tonsillar infection) simulating tuberculosis.

HISTORY.—Early in April, 1927, began to complain of slight pain by night only in the left hip: walked on her toes by day. Improved with rest at home, but as the symptoms did not completely disappear she was sent into a general hospital for X-ray examination, and thence transferred to Bath Orthopædic Hospital (June 15) with diagnosis of 'tuberculosis of hip'.

ON ADMISSION (ten weeks after onset).—Well-nourished and healthy-looking, but slight cough and moderate pyrexia, which persisted on and off for months. Walked with a limp and spared the left lower limb. Only limitation of hip movement was in abduction, which was painful on both sides, but worse on the left, and there only moderate.

X ray of both hips showed no [obvious abnormality, nor did that of lumbar spine, but the sacro-iliac joints were extremely wide and the sacral curve was very marked.

TREATMENT.—On Jones's double hip-abduction frame with traction from July 3 to Sept. 15, i.e., two months; and after that a further two months without traction. Then Thomas walking caliper worn day and night from Nov. 7, and in this she was discharged home on Dec. 2, i.e., after six months' treatment.

PROGRESS.—The spasm in the adductors of the left hip persisted for four months during treatment on the frame, but there was not the rapid wasting which one expects with tuberculosis, and the general condition remained excellent, apart from one attack of pyrexia and abdominal pain, lasting thirty-six hours. No cause was found for this, and culture of urine was sterile.

After discharge from the hospital she failed to report at the clinic, but in June, 1928, was found by the School Medical Officer to have painless effusion in both knees. This failed to clear up with rest at home and Scott's dressing.

ON RE-ADMISSION (Dec. 15, 1928—six months after onset of knee effusion).—Knees had tremendous grating, and much infrapatellar thickening, but the movements were not limited in knees or hips. The only sign of the old hip trouble was $\frac{3}{8}$ in. wasting of the left thigh.

The Wassermann reaction was negative both before and after provocative nov-arsenobillon injection. Skiagrams of the knees showed advanced osteo-arthritic changes. Fluid increased in knees every time she was allowed up.

Tonsils and adenoids were removed on Dec. 20, but there was no obvious effect on joints.

Admitted to general hospital in March, 1930, with mastoid suppuration requiring operation (i.e., three years after onset of symptoms in hip).

Case 2.—J. M., female, age 6 years. Coxa plana simulating early tuberculosis of hip.

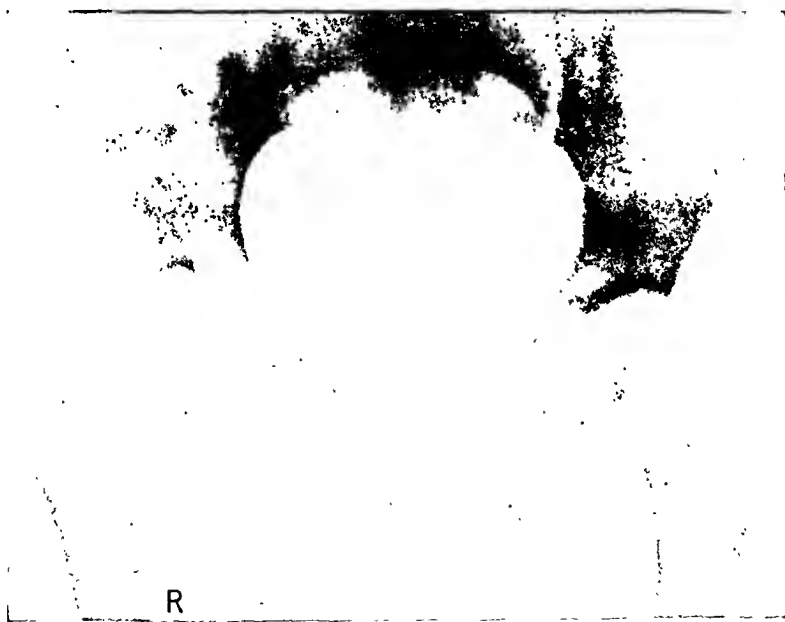


FIG. 36.—Case 2. Coxa plana. Six months after onset of symptoms. Note flattening of head of femur, widening of acetabulum, and increase of joint-interval.

HISTORY.—After 'influenza' in November, 1926, it was noticed that the child limped, and she had pain, which gradually became worse. Diagnosed at first by family doctor as rheumatism, but when it had not cleared up by March, 1927, the patient was sent to an orthopaedic clinic as 'T.B. hip'. Pain and flexion deformity

did not diminish so she was admitted to Bath Orthopædic Hospital on Dec. 10, 1927, i.e., thirteen months after onset.

X rays.—

May, 1927.—Showed typical coxa plana in the right hip; nil in the left (*Fig. 36*).

November, 1927.—Showed extreme flattening and fragmentation of the head of the right femur (*Fig. 37*).

January, 1928.—Shadow of head uniform and one-third outside acetabulum (*Fig. 38*).

Feb. 13, 1928.—Increasing density of head.



FIG. 37.—Case 2. Coxa plana. Thirteen months after onset of symptoms, on admission to hospital. Note fragmentation of head and partial subluxation outwards (not upwards as in tuberculosis).



FIG. 38.—Case 2. Coxa plana. Fourteen months from onset: after one month's recumbency when all symptoms were quiescent and the joint was free except in internal rotation and abduction. Note extreme flattening of head, of which the outer fourth lies outside the acetabulum—density of head more uniform (stage of healing).

For the next eighteen months there was very little change in the X-ray appearance.

ON ADMISSION.—Internal rotation abolished in the right hip, and all other movements were much limited except adduction. The right lower limb showed $\frac{1}{2}$ in. shortening. Pain on forcing hip. Walked with lurch of body to the right.

TREATMENT.—Rest in bed with shoulder straps to keep her recumbent.

PROGRESS.—By Feb. 12, i.e., in two months, the range of the right hip equalled that of the left except that internal rotation was abolished and abduction was 30° instead of 45° . One week later the patient was allowed to begin walking a little (sixteen months from onset).

March, 1928.—High temperature with heavy cold for a few days.

April, 1928.—Range of hips equal, but walks with sway to right: Wassermann negative.

April 28 (eighteen months after onset).—Discharged home; rested a good deal. Limp continued for about six months and then disappeared.

November, 1929.—Half an inch shortening still, and abolition of internal rotation in right hip; otherwise hip and gait appear normal. Skiagram shows old-standing coxa plana. General condition excellent.

Case 3.—W. S., male, age 5 years. Insidious onset of tuberculosis of hip in upper part of acetabulum.

HISTORY.—Vague onset of weakness in left lower limb and pain in left knee early in 1927; then after he knocked his limb on the edge of a chair in May, 1927, the pain was referred to the left groin and he became more lame.

On Nov. 23 he was found by the Tuberculosis Officer to have fixation of the left hip in abduction, slight flexion and eversion, with limitation of other movements, and slight gluteal wasting.

ON ADMISSION.—The boy was admitted to Bath Orthopædic Hospital on Dec. 13 (one year after onset). There was 25° fixed flexion of left hip, and limitation of all movements except external rotation: pain only when the joint was manipulated. The left thigh showed $\frac{3}{4}$ in. wasting, but there was none of the calf. Walked with weight over left lower limb. General condition good, except that he was rather thin. No pyrexia.

X ray.—Suggested some irregularity of lower part of left acetabulum; otherwise two sides identical, and no narrowing of joint-interval.

TREATMENT.—Massage and gentle movements to left lower limb, which increased the range of movement, so that the presence of tubercle seemed unlikely. The boy was accordingly discharged home, and reported regularly each month at the orthopædic clinic near his home.

PROGRESS.—Soon after he was home he began to have occasional night-cries, which became steadily more frequent; the flexion deformity recurred and increased insidiously but steadily, as did the limp, although he continued to keep his body tilted over the left lower limb. By May, 1928, he was unable to walk owing to pain in the groin. Owing to administrative reasons he could not be re-admitted till June, 1928.

ON RE-ADMISSION (June 10—eighteen months after onset).—The left hip allows no movements except adduction: it was still fixed in external rotation, but there was no flexion deformity. One inch wasting of left thigh. Walked with left hip adducted and right knee flexed to compensate. Von Pirquet strongly positive.

X ray.—Showed narrowing of upper part of joint-interval of the left hip and widening of lower part; irregularity of whole acetabulum and subluxation upward of head of femur about $\frac{1}{4}$ in.

PROGRESS.—On Jones's double abduction-frame the hip was abducted and subluxation reduced. The child ran an irregular temperature, and also developed several sore throats with enlarged tonsillar glands.

March 2, 1929.—After nine months on the frame he was treated by Pugh's method of slinging him by the left thigh only, with traction fixed to tilted bed.

Aug. 10.—After five months' slinging, skiagrams showed very little change, so the patient was allowed up in a plaster-spica with patten on the right boot; crutches.

Sept. 6.—Discharged home after fifteen months as an in-patient. This second time at home he was found to be too weak to walk with the crutches. He now slept well, but his appetite was poor, and he got to look progressively paler and sicker.

ON RE-ADMISSION (Jan. 4, 1930—after four months at home in plaster).—The left hip was stiff in good position and painless. The wasting of the left thigh was $2\frac{1}{8}$ in. and of the left calf $1\frac{1}{4}$ in. (formerly none).

X ray.—Showed considerable narrowing of joint-interval and roughness of the head of the femur and of the acetabulum, i.e., destruction of cartilage. No cavities or sequestra in the bone; no subluxation.

General condition poor, with irregular temperature, up every night, even after he was fixed on a double abduction-frame with traction. Sedimentation test was moderately positive, suggesting active disease (three years from onset).

In view of the failure to heal completely and the loss of cartilage, which made

the restoration of a useful joint unlikely, the indication seemed clear for a Hibbs's fusion operation, but it was uncertain whether the child's general condition would stand the operation. However, operation was done in June, 1930, and he was very ill for twelve hours after; then picked up.

Case 4.—H. E., female, age 4 years. Tuberculous focus in neck of femur, involving joint later—signs vague.

HISTORY.—December, 1928, suddenly began to cry day and night with pain in the right leg. This continued for three weeks and seemed relieved by embrocation. Limp ever since. Poor appetite and night-sweats. Kept in bed at home for three weeks in April, 1929, after family doctor had had skiagram taken and recommended admission to orthopædic hospital.

ON ADMISSION.—The child was admitted to Bath Orthopædic Hospital on April 27 (four months from onset). The right hip had some limitation of all movements except hyperextension; no fixed deformity; tenderness over head of right femur. The patient stood and walked with weight over the right lower limb and right hip in external rotation: $\frac{1}{4}$ in. wasting of right thigh; none of calf. Wassermann negative.

X ray.—Showed cavity the size of a shilling in the neck of the right femur; rest nil.

TREATMENT.—Traction to right thigh only by Pugh's method. The child was very difficult to treat as she moved so vigorously that she continually broke the strapping and rubbed her heel into sores.

Swelling in right groin from May 28, 1928, to mid December, 1929, i.e., for seven months.

Jan. 4, 1930.—Plaster-spica applied to right hip and allowed free movement in bed. There was some painless movement in hip, and skiagram showed ossification in the focus with no signs of joint involvement.

This ease with judicious splinting ought to end with full range of hip-joint, but it is hard to say when a focus in the femur is really healed.

Case 5.—K. R., female, age 5 years. Multiple arthritis (probably streptococcal) beginning in one hip and suggesting tuberculosis.

HISTORY.—August, 1924, night-cries with pain in left leg. Could not put it to the ground in the mornings, and after walking a little used to hold it with her hand. Previous to this had had weakness of right leg following four months' recumbency from measles and double pneumonia. Right leg recovered with massage. Always delicate with coughs and colds; had whooping-cough at seven months old, followed by discharging ears for seven weeks.

ON ADMISSION.—The child was admitted to Bath Orthopædic Hospital on Oct. 5 (seven weeks after onset). Left hip flexed and adducted with much spasm; $\frac{1}{2}$ in. shortening of left limb.

X ray.—Showed a little irregularity of the upper part of the left acetabulum, head and neck of left femur; neck shortened. Right hip appeared normal. Night-cries; moderate pyrexia; enlarged iliac glands on both sides; frequently recurring abdominal distension.

TREATMENT.—On Oct. 22 she was put on Jones's double hip-abduction frame.

June 5, 1925.—Hip-spica applied instead of frame.

June 12.—Removal of tonsils and adenoids.

June 26.—Discharged home in plaster. In plaster till March 3, 1926. Plasters changed at clinics. Kept in plaster at home or in hospital till Aug. 27, when a walking caliper was supplied and the patient sent home in this. Plaster re-applied on May 6, 1927 (nine months later), because spasm recurred. Weight-extension to left lower limb on Oct. 24.

May 3, 1928.—Ambulatory hip-splint applied instead of plaster traction.

Sept. 13.—Plaster-gutter to hip.

Nov. 20.—Bed. Thomas's knee-splint applied as there was swelling and pain in left knee.

Feb. 21, 1929.—Abduction frame re-applied, as the left hip had subluxated.

Streptococcal vaccine from recurrent adenoids (Feb. 12) for months; streptococcal vaccine from tooth-socket (Aug. 21) for six months. Hyd. c. cret. gr. j nightly, and colon lavage alternate nights (April 3).

May 3.—Lavage reduced to twice a week; after July 27, once a week.

June 8.—Massage to abdomen and both lower limbs.

June 22.—Radiant heat to left hip.

Aug. 20.—Pugh's method of traction from left thigh only, with boot.

Oct. 4.—Thomas's knee-splint to right limb for spontaneous greenstick fracture of right femur just above knee; reduced under ether.

Nov. 30.—Began walking in gymnasium with head-sling to keep weight off limb. Kept on abduction-frame between treatments till Feb. 3, 1930.

Feb. 15.—Walking-frame alone in ward.

PROGRESS.—Spasm and deformity overcome on frame by June 5, 1925, i.e. in seven months. Febrile attack in June, 1926, a month after she had been re-admitted for swelling of the left foot. Hip stiff at this time and there was iliac induration. Adduction of hip and spasm recurred in May, 1927, i.e. one year later. In September, night-cries and pain recurred (in plaster).

Jan. 4, 1927.—Skiagram showed normal joint-interval in left hip: neck of femur wide and showing two curved spurs from lower surface, i.e., quite unlike tuberculosis.

April 4, 1928.—Skiagram showed roof of left acetabulum oblique and rough; head appeared outside cavity, but not subluxated upward.

June 3.—Von Pirquet positive in eighteen hours.

June 11.—Three provocative tuberculin (Koeli's old tuberculin) injections, the last of 1-10,000 mgrm., negative (four years after onset).

June 12.—No pathological organisms found in stools.

Oct. 13.—Severe pain began in left knee: night-cries.

Oct. 14.—Thickening of left knee and pain on movement: no fluid.

Nov. 21.—Wassermann reaction negative.

Nov. 22.—Swelling of knee gone, but inguinal glands enlarged.

Dec. 21.—Patches over fauces (grew staphylococci only): pyrexia for days.

Jan. 12, 1929.—Throat cleaner: swelling appeared round left hip and increased.

Jan. 29.—Aspiration of the left hip gave some watery, blood-stained fluid, sterile.

Jan. 31.—Adenoids again removed—large mass—grew streptococci, staphylococci, diphtheroids, and *Micrococcus catarrhalis*. Vaccine made from streptococci only, but did little good.

Feb. 9.—Skiagram showed upward subluxation of the head of the left femur; acetabulum irregular.

May.—Vomited after each colon lavage till hyd. c. cret. increased to gr. j.

June 8.—No swelling of any joint: both hips stiff and painless.

June 21.—Pain in left knee recurred after massage and gentle movement.

July 20.—Induration of left calf: no cause found: lasted some days.

July 30.—Pure culture of *Streptococcus viridans* from root of tooth which was extracted under gas.

Aug. 10.—Left knee swollen and hot.

Aug. 22.—No reaction to vaccine from tooth (100 millions in 20 min.) 2 min.

Aug. 28.—Marked local reaction to repetition of same dose of vaccine. Further doses gave no reaction and general condition improved steadily.

Oct. 4.—Woke screaming and found to have greenstick fracture of right femur.

Oct. 21.—Right ankle swollen.

Dec. 21.—Left hip stiff in good position—fibrous ankylosis: 1 in. shortening.

Jan. 4, 1930.—Knees bend only a few degrees: left hip fixed: right very stiff.

March 5.—Pain in right thigh after walking in ward: soon subsided.

Case 6.—A. H., male, age 3 years. Tuberculous hip with abscess and partial destruction of head of femur, ending with full range of movement and apparent regeneration of the head.

HISTORY.—Slipped and fell in October, 1925, since when he complained of pain in the left lower limb and had never walked normally. Early in March, 1926, his

left hip began to swell and pain became worse : skiagram on March 3 is said to have shown erosion of head of femur. Patient sent to Bath Orthopædic Hospital as tuberculous hip.

ON ADMISSION (March 15).—Large elastic swelling over outer aspect of left thigh in its upper third. Movable on the femur, except possibly at a deep neck of attachment. Left hip-joint free except for extreme abduction and external rotation, which were painful. No muscle spasm ; no tenderness on pressure. The patient walked with his body tilted over left lower limb, but otherwise freely and apparently without pain.

X ray.—Showed destruction of inner quadrant of the head of the left femur and adjacent part of the neck. From the region of the small trochanter a uniform shadow extends outward to skin and corresponds to the swelling which is palpable. No other abnormality.

TREATMENT (March 27).—Aspiration of swelling, as it had increased and become adherent to skin : 10 c.c. of thick pus withdrawn. Tubercle bacilli were found present in film of pus, but culture was sterile (Report of Central Laboratory, Bath).

March 28.—Jones's double hip-abduction frame applied—kept on a year.

May 8.—Aspiration again of 10 c.c. of brownish pus.

March 18, 1927.—Plaster-spica applied, including lower ribs and foot (sixteen months from onset, and one year from commencement of treatment). Successive plasters continued till Dec. 18 and again in 1928.

Aug. 18.—Walking with patten on sound side and crutches.

Sept. 26—Nov. 16.—Home in plaster.

Dec. 18—June 16, 1928.—On frame again, as he had a sore on the foot.

June 16—July 9, 1929.—In a series of plaster-spicas, walking about.

July 9.—In Bath Orthopædic Hospital. Plaster valved and gentle massage begun, and active movements.

Aug. 10.—Got up a little in left walking caliper ; recumbent rest of the time without apparatus. (Nearly four years from onset.)

Sept. 6.—Sent home walking in caliper. Kept under observation at County Orthopædic Clinic. Not sent to school.

PROGRESS.—

June 23, 1926.—After three months in hospital and two weeks after second aspiration, had enlarged left iliac glands and swelling in groin, but no fluid.

Sept. 3.—After six months in hospital, and one year from onset, abscess appeared to have absorbed, but five months later a sinus formed at the site of the original aspiration and did not heal for three months.

Winter of 1926.—Much abdominal distension and tendency to constipation.

April, 1927.—Whooping-cough.

June.—Chicken-pox.

Jan. 26, 1929.—A few degrees of movement of left hip in all directions ; some spasm.

April 25.—Same range, but spasm gone. (Three and a half years from onset.)

July 10.—Immediately after removal of plaster—40° active abduction : 15° also of flexion, and external rotation equal to sound side ; other movements limited.

Aug. 3.—One month later, increased to 50° abduction : 35° flexion.

October.—Reported at clinic and had active flexion to 90°.

November.—Flexion still further increased, and internal rotation began.

January, 1930.—Full range of left hip equal in all directions to normal right one. Began to leave off caliper.

February.—Range not diminishing since leaving off caliper.

N.B.—No appreciable shortening ever, but $\frac{1}{4}$ in. true lengthening (Jan. 26, 1929).

X rays.—

Sept. 20, 1926 (six months after admission—one year from onset).—Growth of femur, but proportionate increase of cavity in head and neck.

March 10, 1927.—Cavities closing in.

July 16.—Some regeneration of head of femur. Apparently a sequestrum in neck ; abscess calcified ; joint-interval fairly wide.

Jan. 14, 1928.—Cavity in neck replaced by dense bone ; head flattened. Abscess shadow less obvious (though sinus formed a month later).

Sept. 22 (three years after onset).—Wide, clear joint-interval.

April 20, 1929.—Appearances now resemble coxa plana, except that dense shadow in the neck marks the site of old cavity.

July 16.—Appearances practically unchanged.

Case 7.—L. B., male, age 14 years. Tuberculosis of the right hip with destruction of roof of acetabulum. Spontaneous healing with ankylosis after rupture of abscess (multiple lesions).

HISTORY.—Tuberculous abscess of the left knee. Treated by excision in another hospital and healed in about six months.

1919.—Swelling of the right knee ; healed with limited movement after six months in plaster.

July, 1924.—Sinus formed in region of the right hip and did not heal in spite of four months' recumbency in hospital and seven months more in bed at home.

ON ADMISSION (Oct. 30, 1925—more than a year after onset in right hip).—General condition fair except for pallor. Right hip stiff in 30° flexion and adduction. Right knee subluxated backward and outward : $\frac{3}{4}$ in. true and 1 $\frac{1}{4}$ in. apparent shortening of the right lower limb, in spite of excision of left knee. Three small sinuses on front of right thigh. Left knee ankylosed in good position.

X ray.—Complete destruction of the head of the right femur and subluxation upward of neck, with widening of acetabulum.

TREATMENT.—Jones's double hip-abduction frame one month till deformity corrected. Then plaster-spica in which the boy was sent home after one month more in Bath Orthopædic Hospital.

March 17, 1926.—Frame re-applied, as he had slipped at home after being there six weeks, had run a high temperature, and had to be re-admitted with large abscess in groin (March 3, nearly two years after onset). Aspiration begun on March 17 with 25 c.c., and repeated at first weekly and then daily, with removal of several hundred cubic centimetres (maximum 600 on April 10) till April 18, when abscess burst in perineum and drained freely, finally healing in July, 1927 (more than a year later—three years from onset of disease in hip). Broke down for a few weeks in September, 1928. Series of spica-plasters applied from June 29, 1926, to July 13, 1927.

July 13.—Author's splint applied, and gradually left off in 1929, two years later—i.e., left off at nights in July and completely in November.

RESULT (Feb. 19, 1930).—Firm ankylosis of right hip in good position, with hardly any shortening. X-ray examination suggests that ankylosis is bony, and it did not break down in the six months that have elapsed since the splint was left off. Healing took five and a half years. The right knee has range from straight to right-angle flexion ; is strong. .

Case 8.—V. S., female, age 8 years. Tuberculosis of hip similar to Case 7, but did not heal till fusion operation was done.

HISTORY.—Fell in playground in August, 1922, and was taken to hospital, where no disease was made out, so she was not detained.

April, 1923.—Began to walk with a limp, i.e., nearly one year later. Treated from then for a year in various hospitals by extension, etc.

April, 1924.—Sent home in Thomas splint and supervised at orthopædic clinic.

ON ADMISSION.—The child was admitted to Bath Orthopædic Hospital on Nov. 20, 1925. Very fair hair and freckled skin ; moderately well-nourished. The right hip was stiff in 30° flexion and 15° adduction : 1 $\frac{1}{2}$ in. shortening.

X ray.—Loss of head of the right femur ; neck subluxated up about 1 in. ; acetabulum enlarged and looked 'moth-eaten'.

July 14, 1926 (eight months later).—New bone formation, suggesting ankylosis.

July 30.—Better abduction ; still more new bone.

PROGRESS.—Abscess burst in front of the right thigh on June 27, 1926, and continued to discharge on and off for six months, with no sign of improvement, for when it was dry the temperature rose and the child looked ill, and she was better

each time while it was discharging. Pus sometimes thick, sometimes thin and blood-stained; usually scanty, occasionally profuse. The general condition slowly deteriorated, with pallor, sweating, and loss of appetite, in spite of open-air and sunlight treatment. Hip not stable.

TREATMENT.—Jones's hip-abduction frame from date of admission till Feb. 26, 1927.

OPERATION (Dec. 2, 1926).—Fusion of right hip by sliding graft from great trochanter—a modification of Hibbs's operation (three years after onset). There was no special shock or reaction to operation. The front sinus healed in a few days, and most of the operation wound closed by first intention, but from the back corner of the horseshoe incision some serum drained for many months; the quantity, however, was only a few drops per day, and it was unassociated with any general symptoms. In other words, the child's condition began to improve for the first time within two weeks of the operation, and her progress was steady thereafter.



FIG. 39.—Case 8. Bony ankylosis in right tuberculous hip two years after operation (sliding graft similar to that used in Case 10). Note upward subluxation.

Feb. 26, 1927.—Spica-plaster till Nov. 29, i.e., for nine months.

Nov. 29.—Author's splint applied, but the patient was only allowed to walk with crutches and patten till November, 1928, when bony ankylosis was unmistakable, both clinically and by X rays (Fig. 39). When the splint was first applied a few degrees of movement could still be made out in the hip, though a skiagram (Oct. 24, i.e., ten months after operation) showed much new bone and incorporation of graft with other bones.

Splint gradually left off in 1929. No relapse had occurred by February, 1930.

Case 9.—G. S., female, age 5 years. Tuberculosis of hip failing to become quiescent after five and a half years' conservative treatment, but apparently ankylosing within a few months of fusion operation.

HISTORY.—Sudden onset of acute pain in the right hip in August, 1924, continuous day and night. Treated in another open-air hospital from Aug. 28, where she rapidly improved, though the temperature remained irregular for months, and there was an abscess in the groin, which did not burst.

ON ADMISSION.—The child was admitted to Bath Orthopædic Hospital on Aug. 14, 1925 (one year from onset). She was very fat and fairly well pigmented

Did not look ill. Chronic asthma. The right hip had fibrous ankylosis in slight abduction: 1 in. true shortening.

X ray.—Showed destruction of part of the head of the femur and enlargement of the acetabulum; head wedged closely into cavity, i.e., no dislocation.

July 22, 1926.—Subluxation upward of head about $1\frac{1}{2}$ in.; bone fuzzy.

Sept. 2, 1929.—Complete destruction of head. Great trochanter lay only $\frac{3}{4}$ in. below the anterior superior spine, i.e., subluxation increased.

TREATMENT.—Jones's hip-abduction frame for three months after admission. Plaster-spica from Nov. 26, 1925, to June, 1926; patten; crutches. Thomas walking caliper. June, 1926, to August, 1929, when flexion and adduction had gradually developed in hip, giving $2\frac{1}{2}$ in. apparent shortening.

OPERATION (Oct. 24, 1929).—Fusion of right hip by sliding graft from trochanter (over five years after onset). Cheesy material from the calcified abscess, shown by X rays, escaped when thigh muscles were split. However, primary healing of wound occurred, and the child had little shock or reaction after the operation. Put on hip-frame. No chest complication or recurrence of the asthma during the three months she was in hospital after the operation, although it recurred soon after she went home.

Dec. 21, 1929.—Plaster-spica. Sent home to walk in this for two months.

March, 1930.—Author's hip-splint, when clinically and by X rays firm bony ankylosis seemed to be present, i.e., five months after operation (though it had failed to occur after five years of conservative methods).

Case 10.—M. T., female, age 5 years. Tuberculous hip showing no healing after a year of open-air orthopaedic treatment; rapid improvement after fusion operation.

HISTORY.—In October, 1928, woke with 'cramp' in the right thigh, after which the child limped and got cramps and had occasional pain on movement.



FIG. 40.—*Case 10.* Tuberculous hip after eleven months' orthopaedic treatment. Note loss of joint-interval, subluxation upward of femur, general rarefaction and partial loss of head (not fragmentation as in coxa plana).

November, 1928.—Admitted to open-air orthopaedic hospital (not Bath) and found to have X-ray appearances suggesting tuberculosis of the right hip. Treated for three months on Pugh's extension. Right otitis media for ten days.

January, 1929.—Hip spasm and pain gone, but skiagram showed joint 'woolly'. Put in plaster-spica and got up at the end of January (about four months from onset).

February.—Pain and spasm recurred, so the patient was kept in bed in plaster.

June.—Pain on extreme rotation, and external rotation deformity began.

July.—Put on weight extension to both hips in wide abduction.

Aug. 12.—Put in long right abduction spica in external rotation for journey home.

ON ADMISSION.—The patient was admitted to Bath Orthopædic Hospital, on Sept. 14 (almost one year from onset) in plaster-spica, which was bivalved. She was well nourished and looking well: enlarged tonsils, but no glands. The right hip was stiff in slight abduction and much external rotation; could be moved a little painlessly in all directions. No shortening.

X ray.—Showed considerable destruction of the head of the femur, with subluxation upward from disease of the upper part of the acetabulum; no evidence of abscess, but trabeculae not well defined (*Fig. 40*).

Oct. 16.—Head of femur had almost disappeared; acetabulum perforated; subluxation 1 in.

TREATMENT (Oct. 10).—Tonsils and adenoids removed. Some drowsiness and vomiting for a day after this. No febrile reaction. Picked up quickly.

Oct. 16.—Double hip-abduction frame.

OPERATION (Nov. 14).—Fusion of right hip-joint by sliding graft from great trochanter and part of shaft. There was no infection or fibrosis of the muscles over the joint, but as soon as the graft had been detached in its upper part (root of neck), thin pus escaped from the joint, about 1 oz. in all. The muscles were protected with swabs as much as possible and then swabbed with iodine. When the neck was being rawed to make a bed for the graft, a large portion of the capsule came away in one piece. There was no sign of fibrosis in any region, i.e., no tissue resistance. The abductors were left attached to the graft as a pedicle to ensure its blood-supply. Wound closed without drainage.

PATHOLOGICAL REPORT.—Piece of tissue showed no tubercles, but merely capsular tissue. Guinea-pig killed two months after inoculation showed extensive tuberculosis of spleen, liver, and lung.

PROGRESS.—Retained rectal salines well: was kept quiet with heroin, and showed only moderate initial shock. Pulse kept rapid. On the second day developed great abdominal distension and after a turpentine enema passed only a little flatus and was but partly relieved by cutting all bandages round the trunk. Hyd. e. eret. given four-hourly, and brandy. Slept. On third day began to take food and seemed bright, but in the evening the breath developed the smell of acetone, and the patient looked cyanosed. Alkalis continued and oxygen given for five minutes every hour, which improved the pulse.

Nov. 18.—Immediately after a large movement of the bowels which followed the giving of $\frac{1}{2}$ oz. glycerin per rectum, the patient's pulse suddenly went, and remained feeble for hours, while she had Cheyne-Stokes respiration of irregular type. Gradually improved in the course of the day, and at night temperature rose to 101° , *without rise of pulse-rate*. After this, progress was steady, but the temperature continued to swing for about two weeks, after which it settled in a way it had never done in any previous stage of her illness.

The operation wound appeared to heal by first intention, but one month afterwards a blister formed and a little serum was let out. A small sinus persisted in this region for a long time. There was great swelling in the groin soon after operation, and this took about five weeks to subside entirely.

From this time the general condition improved to an extent it had never done before the operation: there was no more trouble with abdominal distension, and the temperature only occasionally reached 99° , instead of the previous nightly rise.

X rays, Dec. 17 (one month after operation).—Showed graft in good position and no longer any density corresponding to capsule, i.e., no abscess.

Jan. 7, 1930.—New callus appears to unite graft to both femur and ilium (*Figs. 41, 42*).

March 13.—Considerable new bone formation, although no definite trabeculae crossing the line between graft and its bed. No cavities.

Plaster-spica applied to include foot and lower ribs on Jan. 11 and free movement in bed allowed. Another one applied on March 15 for her to walk in.



FIG. 41.—Case 10. One month after fusion operation (sliding graft). Note graft from great trochanter and adjacent piece of shaft slid upward into a bed under periosteum and muscles of ilium.



FIG. 42.—Case 10. Two months after. Note graft from great trochanter and adjacent piece of shaft slid upwards—callus appears to weld both ends of graft to its bed.

March (four months after operation).—No swelling, tenderness, or spasm. Position good. Impossible to say clinically whether the hip is actually ankylosed.

June, 1930 (seven months after operation).—Some movement in hip. No induration or spasm.

Case 11.—D. E., male, age 12 years. Tuberculous hip unhealed six years after onset, in spite of continuous treatment.

HISTORY.—At the age of 6 fell from a saw-mill (1922). Treated immediately at local hospital for eight months, during which time he was found to have tuberculosis of the hip. Sent in plaster to open-air orthopaedic hospital (not Bath), where he was treated by recumbency for a year and eight months. Sent home in celluloid hip-splint, which was changed to plaster by surgeon of local hospital.

August, 1927.—Sent to convalescent home and treated by sunlight and massage, but no splint. Said to have been kicked by another boy, which induced acute pain in the hip.

April, 1928 (six years after onset).—Seen at orthopaedic clinic with flexion deformity and extreme spasm and tenderness of the left hip, in spite of which the parents at first refused admission to hospital.

ON ADMISSION.—The boy was admitted to Bath Orthopædic Hospital on June 16, 1928 (six years from onset). He was well nourished and deeply pigmented, but looked anxious and ill with pain; also flushed, and had moderate pyrexia. The left hip was fixed in right-angled flexion by muscle spasm; extreme tenderness. Fluctuating swelling suggesting abscess over great trochanter. Lordosis.

X ray.—Showed almost complete destruction of the head of the femur, which subluxated upward about 1 in. Dense shadow corresponding to enlarged capsule, and blurring of acetabulum, which was surrounded by irregular, dense bone. Shadows of muscles attached to great trochanter have increased density.

TREATMENT (June 18).—Hip-abduction frame, the left hip being slung in the air at 60° flexion with gentle traction. This relieved symptoms. Aspiration failed to yield pus.

June 20.—Von Pirquet gave strong reaction with lymphatic extension up arm.

July 3.—Weights removed and left limb fixed to frame as flexion overcome (three weeks after admission).

General condition improved rapidly; the boy developed intense pigmentation from exposure to sunlight: the temperature became normal from the middle of July. As conservative measures had failed to heal the disease, it was decided to do a fusion operation before winter came on, while he seemed fit.

OPERATION (Sept 13—six years after onset).—Fusion of left hip by sliding graft from great trochanter—same technique as with Cases 8, 9, and 10. Capsule was tense and burst, letting out thin pus; sequestra and caseous material also removed. No drainage.

PROGRESS.—Woke up within an hour of end of operation, without much initial shock. Vomited a good deal. Four hours after operation the pulse suddenly became irregular and soft, but improved after stimulants and oxygen, etc. Given heroin, gr. $\frac{1}{3}$, for restlessness. Five hours after operation developed Cheyne-Stokes respiration and then suddenly respiration ceased, and though it returned once or twice after artificial respiration was started, it could not be brought back. Heart beat well for about another two hours, during which time artificial respiration was continued, while oxygen and a variety of stimulants were given, including subcutaneous saline, which became absorbed.

It seemed as if the respiratory centre were poisoned by toxins from the hip, or from the contaminated muscles.

NOTE.—Cases 8–11 were prepared for operation by giving glucose and pot. cit. for at least a week beforehand. They were given rectal salines containing these substances before they left the theatre, and were treated by warmth, raising the foot of the bed, and heroin before they should wake. They were all put on Jones's double hip-abduction frame immediately at the end of the operation, and all four were operated on by the writer, so that differences in progress must be regarded as due, not to differences of technique or treatment, but to different qualities in their own resistances to the toxins of tuberculosis. It is this which it would be so valuable to foretell, if one could do so, but these records suggest that it is an extremely difficult matter, and that we have at present no reliable guide. This places the fusion operation in the position of a gamble at present. Yet it is evident from these records also that conservative treatment is just as speculative an affair, and though a fatal issue may not ensue from it in so dramatic a manner as after operation, yet so long as the hip contains living tubercle bacilli, the patient risks a sudden termination from meningitis at any moment.

A BACTERIOLOGICAL STUDY OF FIFTY CASES OF CHOLECYSTECTOMY WITH SPECIAL REFERENCE TO ANAEROBIC INFECTIONS.

By G. GORDON-TAYLOR AND LIONEL E. H. WHITBY,
MIDDLESEX HOSPITAL, LONDON.

WHEREAS it is universally accepted that bacterial infection is the probable cause of most cases of cholelithiasis, it is by no means certain that the particular bacteria usually cited as the causal agents are of necessity the primary pathogenic organisms, nor has the route of primary infection been definitely established: blood-stream, lymphatics, and bile-passages have all been regarded as the possible channel by which the bacteria have reached the gall-bladder.

Table I.—BACTERIA ISOLATED FROM GALL-BLADDER WALL, FROM
BILE, AND FROM STONES.

CASES		ANALYSIS
All three tissues infected	13	6 <i>B. coli</i> ; 3 <i>Str. faecalis</i> ; 2 <i>Sta. albus</i> ; 2 <i>B. welchii</i>
Wall and bile only	3	1 <i>Str. faecalis</i> ; 1 <i>Sta. albus</i> ; 1 <i>B. welchii</i>
Wall only	25	3 <i>B. coli</i> ; 1 <i>B. paratyphosus B</i> ; 7 <i>Str. faecalis</i> ; 3 <i>Sta. albus</i> ; 1 <i>Sta. aureus</i> ; * 3 <i>B. welchii</i> ; † 7 mixed.
Stone only	1	1 <i>B. welchii</i>
All three tissues sterile	8	
	50	* One case, no stone—'mud' only. † Mixed infection: 2 <i>B. coli</i> + <i>B. welchii</i> , 2 <i>B. coli</i> + <i>Str. faecalis</i> , 2 <i>B. coli</i> + <i>Sta. albus</i> , 1 <i>B. acidilactici</i> + <i>Str. faecalis</i>

Analysis of Incidence of Various Bacteria in Fifty Cases, including Mixed Infections.—Sterile 8; *B. coli* 15; *B. welchii* 9; *Str. faecalis* 14; *Sta. albus* 8; *Sta. aureus* 1; *B. paratyphosus B* 1; *B. acidilactici* 1.

Rosenow (1916), Wilkie (1928), and others have stressed the part played by streptococci in the production of gall-bladder infections, basing their evidence on bacteriological findings obtained from clinical material as well as on animal experiments. As a result, one modern view of the pathology of cholecystitis is that the infection arises most commonly from blood-borne bacterial emboli which set up an intramural infection of the gall-bladder. Such emboli are said to arise from a remote septic focus, and much prominence is given to apical dental infections which are so commonly streptococcal. Tables II and III show clearly that infection of the wall of the gall-bladder is much more common than is that of the fluid contents. The older view that gall-stones and cholecystitis arise from the penetration of intestinal bacteria into the lumen of the gall-bladder has thus been superseded, and with the abandonment of the old view there is a tendency perhaps to over-emphasize the part played by streptococci, to relegate the intestinal bacteria

to a secondary place, and to regard the latter as terminal infections in cases of lithiasis rather than as primary causal agents.

Yet the frequent incidence of intestinal bacteria in gall-bladder infections cannot be denied, and it is indisputable that the anatomical position of the

Table II.—FLUID CONTENTS OF GALL-BLADDER.

INVESTIGATORS	NO. OF CASES	INFECTED	STERILE	REMARKS
Present series ..	50	16	34	6 <i>B. coli</i> ; 4 streptococci; 3 <i>B. welchii</i> ; other bacteria 3
Blalock (1924) ..	270	157	113	77 <i>B. coli</i> ; 29 <i>B. typhosus</i> ; 8 streptococci; 6 <i>B. coli</i> and streptococci; other bacteria 37
Drennen (1922)	100	19	81	12 <i>B. coli</i> ; 2 streptococci; 4 <i>Sta. aureus</i> ; other bacteria 1
Friesleben (1928)	132	62	70	37 <i>B. coli</i> ; 14 streptococci; other bacteria 11
Hartmann (1903)	46	36	10	25 <i>B. coli</i> ; 3 streptococci; 4 staphylococci; other bacteria 4
Illingworth (1927)	100	40	60	20 <i>B. coli</i> ; 16 streptococci; other bacteria 4
Johnson (1925) ..	100	32	68	18 <i>B. coli</i> ; 3 streptococci; 7 <i>Sta. aureus</i> ; other bacteria 4
Judd, Mentzer and Parkhill (1927)	193	28	165	5 <i>B. coli</i> ; 6 streptococci; 1 <i>B. welchii</i> ; 7 <i>B. coli</i> and streptococci; 2 streptococci and staphylococci; other bacteria 7
Rosenow (1916)	29	16	13	4 <i>B. coli</i> ; 5 streptococci and <i>B. coli</i> ; other bacteria 7
Rovsing (1923) ..	530	216	314	—
Wilkie (1928) ..	50	6	44	3 <i>B. coli</i> ; 2 streptococci; 1 <i>B. welchii</i>
Total ..	1600	628 (39%)	972 (61%)	

Table III.—WALL OF THE GALL-BLADDER

INVESTIGATORS	NO. OF CASES	INFECTED	STERILE	REMARKS
Present series ..	50	41	9	9 <i>B. coli</i> ; 11 streptococci; 3 <i>B. welchii</i> ; 5 <i>B. welchii</i> with other organisms; 13 other bacteria
Friesleben (1928)	96	73	23	27 <i>B. coli</i> ; 13 streptococci; 31 staphylococci; other bacteria 2
Huntemuller (1924)	150	150	0	—
Illingworth (1927)	100	62	38	17 <i>B. coli</i> ; 34 streptococci; 5 streptococci and <i>B. coli</i> ; other bacteria 6
Judd, Mentzer and Parkhill (1927)	200	98	102	9 <i>B. coli</i> ; 26 streptococci; 20 staphylococci; 2 <i>B. welchii</i> ; other bacteria 41
Rosenow (1916)	32	27	5	1 <i>B. coli</i> ; 10 streptococci; 8 streptococci and <i>B. coli</i> ; 6 <i>B. welchii</i> with other bacteria; 1 <i>B. welchii</i> ; other bacteria 1
Wilkie (1928) ..	50	6	44	3 <i>B. coli</i> ; 1 <i>B. welchii</i> ; 2 streptococci
Total ..	678	457 (67%)	221 (33%)	(N.B.—Cystic gland: 45 positive cultures out of 50 cases; 43 streptococci)

liver and gall-bladder definitely favours infection from the intestinal tract. Furthermore, the activities of intestinal bacteria are almost invariably accompanied by acid production—a fact which Kramer (1907) and others

have shown to be of great importance for the precipitation of cholesterol. While it must be admitted that cholecystitis is usually due to an intramural infection, and that the latter is often streptococcal, yet on bacteriological evidence, as well as on considerations of probability, it would seem that not only are the intestinal bacteria equally important, but that, on account of their acidogenic power, they are particularly liable to be associated with lithiasis.

To the usual list of intestinal bacteria so frequently found in gall-bladder infections, we would add one other, the *Bacillus welchii*, an organism with remarkable acidogenic properties. *B. welchii* has been detected by us in some portion of the excised gall-bladder in no fewer than 9 out of 50 consecutive cases (*Table I*). The frequency of the finding, as compared with that of other investigators, is undoubtedly due to the fact that, as a routine, we made

Table IV.—GALL-STONES.

INVESTIGATORS	NO. OF CASES	INFECTED	STERILE	REMARKS
Present series ..	50	14	36	6 <i>B. coli</i> ; 3 streptococci; 3 <i>B. welchii</i> ; 2 staphylococci
Present series (post-mortem)	31	5	26	3 <i>B. welchii</i> ; 2 streptococci
Gilbert (1898) ..	70	23	47	—
Illingworth (1927)	23	7	16	3 <i>B. coli</i> ; 2 streptococci; 2 staphylococci
Judd, Mentzer and Parkhill (1927)	67	24	43	2 <i>B. coli</i> ; 6 streptococci; 2 sporing bacilli; other bacteria 14
Rosenow (1916)	62	20	33	17 streptococci; 1 <i>B. coli</i> ; 3 streptococci and <i>B. coli</i> ; 1 <i>B. welchii</i> ; 6 <i>B. welchii</i> with other bacteria; other bacteria 1
Total ..	303	102 (53%)	201 (66%)	

anaerobic as well as aerobic cultivations. In fact *Tables II, III, and IV*, though emphasizing the frequency of infections with intestinal bacteria, give no indication of the incidence of anaerobic infections, since the latter, in so many instances, were not looked for.

The present series of fifty consecutive cases was, with a single exception, carried out on fresh material derived from subjects appearing for cholecystectomy in the hospital and private practice of one of us (G. G.-T.).

TECHNIQUE.

The unopened gall-bladder, wrapped in sterile gauze, has been sent straight to the laboratory and there dealt with in the following manner:—

After searing the external surface of the gall-bladder, the organ was incised with a sterile scalpel; through the incision some of the fluid contents were extracted. About 0.5 c.c. was planted on to agar, and into glucose broth, and incubated aerobically; a similar amount was incubated, under anaerobic conditions, in meat broth and litmus milk. Stained smears of the bile were examined at the same time. A piece of gall-bladder wall was washed in saline, and parts of it were implanted in glucose broth and meat broth for aerobic and anaerobic incubation. Stones were placed in running water and

washed continuously for twenty-four hours. Thereafter they were placed in 5 per cent carbolic acid for half an hour, transferred to absolute alcohol for a few minutes, and then removed, the excess alcohol being burnt off. The whole stone was then incubated in glucose broth aerobically for forty-eight hours, and if any growth occurred the sterilizing process for the external surface was repeated. If the aerobic culture was sterile, the whole stone was incubated anaerobically in meat broth for forty-eight hours. If growth occurred, the sterilization process was repeated, in some cases several times, until anaerobic culture was sterile. Thereafter the stone was crushed in a mortar, and the crushings were incubated aerobically in glucose broth, and anaerobically in meat broth and litmus milk. This method assures almost with certainty that such organisms as develop are derived from the inner parts of the stone.

B. WELCHII INFECTIONS.

Clinical Notes.—The two cases in which *B. welchii* was obtained in pure culture from the gall-bladder wall, the bile, and the central parts of the gall-stones were acute gangrenous cases. In each the bacilli were so numerous that they could be easily found in a stained smear of the purulent bile fluid. In one of these cases there must have been some secondary change in the stomach wall, for a severe hæmatemesis occurred during the induction of anaesthesia and nearly produced a fatal catastrophe on the operating-table. Furthermore, as soon as the abdomen was opened a copious hæmorrhagic effusion poured from the wound and occasioned still further alarm; the gall-bladder was removed, but the patient died twenty-four hours later despite the administration of *B. welchii* serum. The other case, though desperately ill at the time of operation, made a good and quick recovery, and we attribute the rapid subsidence of toxæmia in part to antiserum.

Of the five cases in which *B. welchii* was isolated from the gall-bladder wall, three were acute cases with hæmorrhagic oedema of the gall-bladder, of which the contents were partially purulent. Antiserum was administered to one of these and appeared to hasten recovery. The two other cases, together with that in which the organism was isolated from the bile as well as the tissue, were all subacute, with a slightly inflamed gall-bladder.

The one case* in which *B. welchii* was isolated from the stone only was an ordinary chronic cholecystitis with a small fibrotic gall-bladder and one large stone.

Our experience suggests that *B. welchii* is associated mainly with the acute forms of cholecystitis, and it is reasonable to inquire whether the organism is a relic of a previous infection from which lithiasis resulted, or whether it is merely a secondary invader, in a case of acute cholecystitis, contributing materially to the gangrene of the gall-bladder. Opinion on these points can be only speculative.

Significance of *B. welchii* Infections.—Standard works on gall-bladder disease pay but little attention to anaerobic infections, and attribute to

* This patient had also exhibited achylia.

B. welchii only a contributory power in the causation of gangrenous cholecystitis. Routine investigators such as Rosenow (1916) and Judd, Mentzer, and Parkhill (1927) have regarded the finding of *B. welchii* in a proportion of cases as merely incidental. And furthermore, actual cases of acute cholecystitis where the infection was obviously due to *B. welchii* have been regarded as surgical rarities (Cottam, 1917; Hallé and Marquezy, 1922; Lereboullet, 1922; Kirchmayr, 1925; Gould and Whitby, 1927).

Gall-stones may arise from a comparatively mild inflammation of the gall-bladder. Once formed, however, stones dispose not only to fresh infection but also to a more acute cholecystitis as well as to a persistence of the original infection. Yet the long duration of the inflammatory process results often in the gall-bladder and its contents being found to be sterile when bacteriologically examined. Thus the stones, being but a monument to the primary infection, have become the tomb of the causal bacteria.

Anaerobes, owing to their sporing powers, have obviously a greater chance of surviving interment in a gall-stone than have non-sporing bacteria. This must be the explanation for our finding *B. welchii* in the central portions of nearly 13 per cent of gall-stones (*Table IV*) obtained from post-mortem cases—cases from which the gall-stones were taken merely because they were present and not because of a history pointing to gall-bladder infection. This finding agrees with that of Rosenow (*Table IV*) and appears to us to be some evidence for believing *B. welchii* to be a possible cause of a primary gall-bladder infection. Furthermore, the gall-bladder is an organ which may become congested from the pressure of a loaded colon, from ptosis, or from the internal pressure of its contents; such congestion is associated with anoxæmia, a condition favourable for the multiplication of *B. welchii*. It is well known that *B. welchii* is pre-eminently an organism of dead or dying tissue, so that its presence in a frankly gangrenous gall-bladder is to be expected rather than wondered at. As to the transition from a relatively quiescent inflammation to one of acute gangrene, Fildes (1927) has shown that anoxæmia due to congestion is the ideal condition for the activation of quiescent anaerobic spores.

And with regard to the streptococci that were isolated from our cases, not one was of the hæmolytic variety. As far as the rather indefinite fermentation reactions may be trusted, the majority belonged to the *faecalis* type—a type well known to be resistant to heat, to drying, and to antiseptics—in fact an organism which, like *B. welchii*, would have more chance of surviving long desiccation within a gall-stone than less hardy bacteria.

As to the route of infection, the most reasonable anatomical explanation is via the portal system to the liver, and thence by the periportal lymphatics. It is well known that intestinal bacteria invade the blood-stream just before death, and there is some evidence that the phenomenon is not always agonal. Thus Berg, Zan, and Jobling (1927), experimenting with dogs, have shown that prolonged starvation results in the appearance of *B. welchii* in the liver, whilst it is the custom, when bleeding horses for a supply of serum, to deny the animals rough food for some hours before bleeding; unless this precaution against intestinal irritation is taken, the serum is frequently found to be contaminated with intestinal anaerobes. Undoubtedly the factors which facilitate

the entry of bacteria into the portal circulation are inflammation and infection of the intestinal mucosa. To these may be added in less drastic circumstances, the phenomenon of achlorhydria which, as Davidson (1928) and others have shown, is associated with an abundant bacterial flora from the duodenum downwards.

We would urge that a review of the statistics of the literature as summarized in *Tables II, III, and IV* still places the intestinal bacteria as the commonest organisms to be found in gall-bladder infections, and that the prominence we have given to *B. welchii* in this paper serves still more to emphasize the fact.

SUMMARY.

1. Intestinal bacteria are the most frequent organisms in gall-bladder infections.

2. *B. welchii* is usually, but not invariably, associated with the acute form of cholecystitis.

3. *B. welchii* is more commonly found in gall-bladder infections and in gall-stones than has hitherto been believed: in nearly 9 per cent of a series of gall-bladders removed by operation was this organism found.

4. *B. welchii* was found in the centre of 13 per cent of gall-stones obtained from the post-mortem room.

One of us (L. E. H. W.) is indebted to the Medical Research Council for a grant for the performance of this work.

REFERENCES.

- BERG, B. N., ZAN, Z. D., and JOBLING, J. W., *Proc. Soc. Exper. Biol. and Med.*, 1927, xxiv, 433.
 BLALOCK, A., *Johns Hopkins Hosp. Bull.*, xxxv, 391.
 COTTAM, W. G., *Surg. Gynecol. and Obst.*, 1917, xxv, 192.
 DAVIDSON, L. S. P., *Jour. Pathol. and Bacteriol.*, 1928, xxxi, 557.
 DRENEN, J. G., *Ann. of Surg.*, 1922, lxxvi, 482.
 FILDES, P., *Brit. Jour. Exper. Pathol.*, 1927, viii, 387.
 FRIESLEBEN, M., *Münch. med. Woch.*, 1928, lxxv, 81.
 GILBERT, A., *Arch. gén. de Méd.*, 1898, clxxxii, 257.
 GOULD, E. P., and WHITBY, L. E. H., *Brit. Jour. Surg.*, 1927, xiv, 646.
 HALLÉ and MARQUEZY, *Bull. et Mém. Soc. Hôp. de Paris*, 1922, xli, 13.
 HARTMANN, O., *Deut. Zeits. f. Chir.*, 1903, lxxviii, 207.
 HUNTEMULLER, von, *Klin. Woch.*, 1924, lii, 349.
 ILLINGWORTH, C. F. W., *Brit. Jour. Surg.*, 1927, xv, 221.
 JOHNSON, W. O., *Amer. Jour. Med. Sci.*, 1925, clxx, 181.
 JUDN, E. S., MENTZER, S. H., and PARKHILL, E., *Ibid.*, 1927, clxiii, 16.
 KIRCHMAYR, *Zentralb. f. Chir.*, 1925, lii, 1522.
 KRAMER, S. P., *Jour. of Exper. Med.*, 1907, ix, 319.
 LEROULLETT, M. P., *Bull. et Mém. Soc. Hôp. de Paris*, 1922, xli, 257.
 ROSENOW, E. C., *Jour. of Infect. Dis.*, 1916, xxix, 527.
 ROYSENG, T., *Acta Chir. Scand.*, 1923, lvi, 103.
 WILKIE, A. L., *Brit. Jour. Surg.*, 1928, xv, 450.

CARCINOMA OF THE DUODENUM.

By J. EWART SCHOFIELD,

ASSISTANT SURGEON TO THE GREAT WESTERN HOSPITAL, SWINDON.

THAT carcinoma of the duodenum is an extremely rare condition is strongly emphasized by the fact that little or no attention is paid to it by medical and surgical text-books. In view of the frequency of carcinoma of the stomach, this rarity is all the more surprising, and the difference between the two organs with regard to the incidence of carcinoma has given rise to much discussion. In cases of carcinoma of the duodenum it is not unlikely that a wrong diagnosis will be made, and the condition may not even be considered in the differential diagnosis. At laparotomy one may be confronted suddenly with the disease, and thus be forced to undertake its treatment without sufficient time in which to consider the best method to adopt. The frequency of carcinoma of the duodenum is shown in *Table I*.

Table I.—FREQUENCY OF CARCINOMA OF THE DUODENUM.

AUTHOR			AUTOPSIES	CANCER OF DUODENUM
Schlesinger ¹ (Vienna)	42,000	7
Perry and Shaw ² (Guy's Hospital)	17,652	4
W. S. Fenwick ³ (London Hospital)	19,500	18
Maydl ⁴ (Vienna)	20,480	2
Nothnagel ⁵ (Vienna)	31,358	5

The disease, however, is probably more common than these figures suggest, since the duodenum does not always escape in pyloric cancer. Microscopical examination has shown that the duodenum is sometimes involved in pyloric cancer, but only for a few centimetres from the pylorus, and Nagel⁶ quotes 14 cases of his own of cancer of the pylorus in which the duodenum was macroscopically involved in three, but which on microscopical examination showed carcinoma to be present in five. Other writers, according to Nagel, found the duodenum to be involved microscopically as shown in *Table II*.

Table II.—FREQUENCY OF DUODENAL INVOLVEMENT IN CANCER OF PYLORUS.

AUTHOR			CANCER OF PYLORUS	DUODENUM MICROSCOPICALLY INVOLVED
Konyetzny	54	23
Brinton	125	10
Maragliano	12	2
Borrmann	63	20

The percentages are surprisingly high, and although it appears that the pylorus is not a barrier to the spread of the disease, nevertheless the duodenum is highly resistant to carcinoma.

It is interesting at this stage to consider the relative incidence of carcinoma of the intestine. That the large bowel is the most frequent site of intestinal carcinoma is well known, and Eusterman, Berkman, and Swan⁷ state that in cases of intestinal cancer 3 per cent only occur in the small intestine. With regard to the regional incidence, the duodenum appears to be the most commonly affected, the ileum to a less extent, and cancer of the jejunum is practically unknown. Of 12 cases of small intestinal cancer, Kohler⁸ found 9 to be present in the duodenum, and Deaver and Ravadin⁹ state that "The relative proportion of cancer of the duodenum on the one hand to cancer of the jejunum and ileum on the other, is 47.7 per cent to 52.2 per cent." Jefferson¹⁰ found that the duodenum was affected in 34 out of 71 cases of small-intestinal carcinoma—that is, 48 per cent—and states, "Considering the shortness of the duodenum, it is evident that, inch for inch, the duodenum is more liable to carcinoma than the rest of the small intestine."

DISTRIBUTION OF DUODENAL CANCER.

Dividing the duodenum for descriptive purposes into supra-ampullary, ampullary, and infra-ampullary regions, as suggested by Sherren and quoted by Sir John Bland-Sutton,¹¹ *Table III* shows the frequency with which they are affected:—

Table III.—DISTRIBUTION OF DUODENAL CANCER.

AUTHOR	SUPRA-AMPULLARY	AMPULLARY	INFRA-AMPULLARY
Rolleston ¹²	8	24	3
Geiser ¹³ ..	11	51	9
Fenwick ..	11	29	7

It will be noticed that by far the greater number of duodenal cancers occur in the ampullary region, but it is probable that this high percentage is due to difficulty in distinguishing cancer of the ampulla of Vater from primary duodenal cancer. According to Nothnagel, duodenal cancer tends to encircle or extend along the intestine, whilst cancer of the ampulla of Vater is sharply circumscribed about the ampulla. Even in advanced cases of duodenal cancer the occlusion may be incomplete, jaundice may be absent, and gastric symptoms prominent. In cancer of the ampulla, jaundice is an early and persistent symptom.

ETIOLOGY.

The causation of duodenal cancer has been a much debated subject, especially with regard to the predisposition of duodenal ulcer to carcinoma. Many theories have been put forward. By some it was thought that the condition arose in aberrant stomach glands. By others that the primary site was in pancreatic rests, which are known to occur especially in the first part

of the duodenum. Orth¹⁴ thought that the cancer had its origin in Brunner's glands.

The question of cancer arising in a duodenal ulcer is of some interest. In the stomach, Walton¹⁵ thinks that 20 per cent of carcinomata are preceded by ulcer. The evidence concerning the duodenum is as follows: A case of colloid cancer occurring in a duodenal ulcer, the cicatrix of which was seen, is mentioned by Letulle.¹⁶ The only case of malignant disease of the duodenum seen by Bland-Sutton was in a man of 60 years in whom a supra-ampullary ulcer had turned sarcomatous; there was a polypoid mass in the duodenum with a well-marked scar opposite the pylorus. A case of carcinoma of the duodenum occurring not in but just below a duodenal ulcer is recorded by Dewes and Morse.¹⁷ Jefferson has collected thirty cases, including five of Perry and Shaw's, and five of Nattan-Larrier's, in which ulcer is supposed to have preceded carcinomata, but he thinks that these cases are doubtful, and adds, "A causal relationship between simple ulcer and cancer is difficult to establish in the case of the duodenum." It is a remarkable fact that so few cases of duodenal cancer occur in view of the frequency of duodenal ulcer. Furthermore, cancer of the duodenum is most often found in the region of the ampulla, whereas ulcer is practically confined to the supra-ampullary region as shown by Collin,¹⁸ who found that 242 of 262 cases of duodenal ulcer were in the supra-ampullary region of the duodenum.

SYMPTOMATOLOGY.

The symptoms naturally depend on the site of the growth.

1. **Supra-ampullary.**—The symptoms of growth in this region closely resemble those of pyloric cancer.

2. **Ampullary.**—Here the symptoms are mainly those of obstruction of the common bile-duct, and it requires only a small growth to produce a serious condition of the patient. Carcinoma above or below the ampulla may progress until the bowel is occluded without giving rise to serious symptoms; but a small ampullary growth produces rapid emaciation and death. Jaundice with enlargement of the liver and gall-bladder is usually an early symptom. Pain is not a marked feature, but wasting occurs early in the disease owing to the obstruction to the flow of the pancreatic juice. Vomiting is usually present, but hæmatemesis seldom occurs. Dilatation of the biliary and pancreatic ducts is often present, and Ewing¹⁹ states that fat necrosis has been observed by Krause, Gerster, and Geiser. Carcinoma of the head of the pancreas, and stone in the common bile-duct, are usually considered in the differential diagnosis, but ampullary cancer would probably not be taken into account. The most likely diagnosis would be carcinoma of the head of the pancreas, and it may be urged that all cases of obstructive jaundice except those due to metastases, and those in which the operative risk is too great, should be subjected to laparotomy.

3. **Infra-ampullary.**—Nausea and indigestion are early symptoms, followed by copious vomiting, the vomit containing large quantities of bile. Rapid emaciation follows. The stomach and duodenum proximal to the growth become enormously dilated.

TREATMENT.

When confronted by a case of duodenal cancer, the treatment is determined by the site, the size, fixity, and metastases, which occur late. Many writers lay stress on the fact that secondary deposits appear late in the disease, and Outerbridge²⁰ reports 110 cases in which metastases were present in only 25.

Supra-ampullary Cancer.—This is treated on the same lines as pyloric cancer.

Ampullary Cancer.—With regard to the more common ampullary cancer, two main methods of treatment are open to the surgeon :—

1. **PALLIATIVE TREATMENT.**—In this method the jaundice is relieved either by cholecystostomy or anastomosis of the gall-bladder to the stomach or first part of the duodenum. Internal fistula is usually preferred, since it gives the patient the benefit of the bile, and avoids a copious discharge of bile on to the surface of the abdomen, which is distressing to the patient. Vomiting may be relieved by gastro-enterostomy.

2. **RADICAL TREATMENT.**—

Excision of growth, preferably by the transduodenal route, may be employed when the growth does not extend far from the ampulla, but the operation entails considerable risk to the patient, and several technical points may have to be dealt with. It may be necessary to reimplant the common bile-duct and the duct of Wirsung into the duodenum. In a series of 59 cases of ampullary carcinoma subjected to operation collected by Cohen and Colp,²¹ 53 were treated by excision, 50 by the transduodenal, and 3 by the retroduodenal route. Of these 53 cases, only 18 survived the operation and its immediate after-effects; 9 are reported alive after twelve months; and 5 after two years. The following table shows the cause of death.

Hæmorrhage	7	Fistula	2
Peritonitis	2	Recurrence	13
Shock	2	Other causes	9

Resection of duodenum may be performed in cases where the growth has extended well away from the ampulla. This is a tremendous operation, and Cohen and Colp report that after a careful examination of the literature only the cases of Halstead, Kausch, Herschel, and Tenani survived the operation.

It therefore appears that radical treatment is attended by so high a mortality as to render the procedure unwarranted, and palliative treatment offers only temporary relief.

Infra-ampullary Cancer.—Resection is probably the best operation. Panchet and Luquet²² have reported a case of carcinoma of the fourth part of the duodenum in a woman 61 years of age. With a history of three months' abdominal pain, and X rays showing the third part of the duodenum to be almost as large as the stomach, a diagnosis of pressure on the fourth part of the duodenum by superior mesenteric vessels was made. The third and fourth parts of the duodenum were mobilized and resected, and the proximal part was sutured to the jejunum. The patient later returned to work.

CASE REPORT.

HISTORY.—A married woman, age 35 years, was admitted under my care to the Hackney Hospital on March 28, 1929. She gave a history of epigastric pain over a period of several months, not severe, no suggestion of colic, and not related to the taking of food. Vomiting had occurred on and off for three months, but had been persistent during the last fortnight. She had been jaundiced for about two months, and volunteered the statement that the depth of the jaundice had varied from day to day. She complained of severe wasting, and of feeling very weak and tired.

ON EXAMINATION.—The patient looked ill and emaciated and was running an intermittent temperature. Jaundice was marked, and there were several abrasions of the skin due to scratching. The tongue was dry and furred. Pyorrhœa was present. A deformity of the soft palate suggested a healed perforation, but there were no other signs of syphilis, and the Wassermann reaction was negative. There was a history of a severe attack of diphtheria in childhood. The abdomen was wasted, and there was a tumour in the right upper abdomen, extending as far as the umbilicus. This was thought to be liver, but the gall-bladder could not be felt as a separate swelling. The urine contained bile, and the faeces, although pale, were not clay-coloured. The case received various diagnoses, but carcinoma of the head of the pancreas was the most popular.



FIG. 43.—Duodenum opened, edges retracted, exposing malignant ulcer on postero-medial wall, encroaching proximally on ampulla.

OPERATION (April 16).—For two days previous to the operation calcium chloride and hæmostatic serum were administered. The abdomen was opened through a right upper paramedian incision under a general anæsthetic. There was no 'free fluid' present in the peritoneal cavity. The liver was enlarged, and the gall-bladder distended. An oval swelling was felt in the duodenum in the region of the ampulla of Vater. To ascertain the nature of the tumour the anterior surface of the duodenum was opened, and the interior exposed. A growth, about one and a quarter inches long, was lying on the postero-medial wall of the duodenum; the proximal end encroaching on the ampulla was causing partial obstruction to the flow of bile (Fig. 43). The edges of the ulcer were raised and friable. A probe passed easily up the common bile-duct, and when withdrawn there appeared a copious flow of bile. There were no secondary deposits found, and a diagnosis of infra-ampullary carcinoma involving the ampulla of Vater was made. The growth was thought to be infra-

ampullary rather than ampullary because the shape suggested growth from below, and the history of jaundice was comparatively recent. A piece of the edge of the growth was removed for section and the duodenum closed. The condition of the patient was poor, so a palliative operation was decided upon. Cholecystoduodenostomy and posterior gastro-enterostomy were performed and the abdomen closed. The patient made good progress, all pain disappeared, vomiting ceased, the jaundice faded, and weight increased rapidly.

MICROSCOPICAL EXAMINATION.—Histological examination of the piece removed for section showed 'adenocarcinoma' (Fig. 44).

Six weeks later I decided to attempt a radical cure of the disease, not by excision or resection, but by the implantation of radon seeds. The patient was doing so well that I had the greatest difficulty in persuading her to undergo a second operation.

SECOND OPERATION (May 28).—Under a general anæsthetic the abdominal scar was excised. There were many adhesions in the upper abdomen, possibly due to bile spilt at the first operation, and the duodenum was reached after an effort. The liver was almost normal in size, but the gall-bladder was hidden in a mass of adhesions. There were still no signs of secondary deposits. The anterior surface of the duodenum was re-opened, this time in a transverse direction, and the growth exposed. Although six weeks had elapsed, there was very little increase in the size of the growth. Since the tumour was on the postero-medial wall of the duodenum, adjacent to the head of the pancreas, and since adhesions were numerous, it was impossible to introduce the radon seeds without opening the duodenum. Eight radon seeds of 1.6 mc. each were introduced, seven at regular intervals around the growth, and one into its centre. The duodenum was closed with difficulty owing to adhesions binding it down to the posterior abdominal wall.

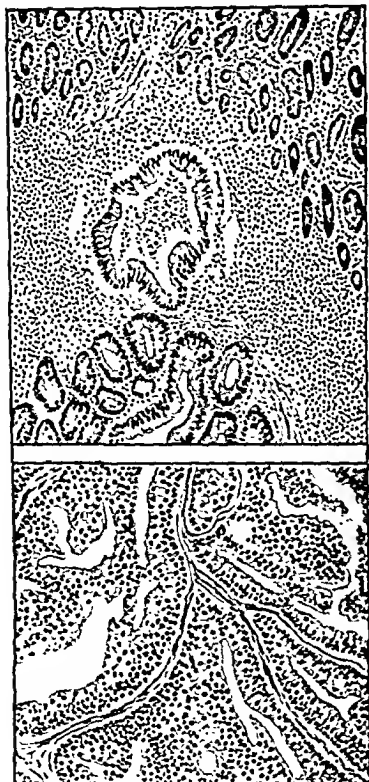


FIG. 44.—The lower figure shows a section of piece removed at first operation showing columnar-celled carcinoma of duodenum, alveoli being lined in parts by a layer of columnar cells, in others the cells proliferate irregularly and partly fill the lumen. Stroma scanty. Above, normal duodenum at edge of growth. ($\times 65$.)

The patient did well until the fifth day after the operation, when a duodenal fistula developed. This was most disappointing, and resulted in her death on June 26, 1929, twenty-nine days after operation. To open the duodenum again at the second operation was tempting Providence, and that the leakage was due to difficulty in closing it again I have little doubt, since the duodenum

is notoriously prone to leak: but it was suggested by my colleague, Mr. J. C. Gillies, that the healing of the suture line may have been interfered with by the action of the radon seeds.

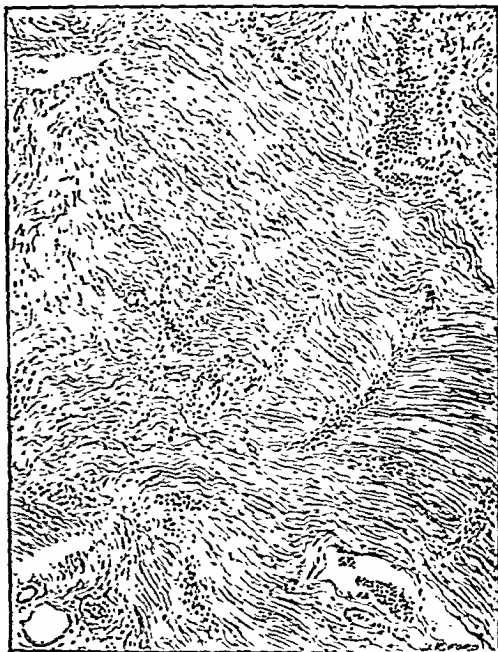


FIG. 45.—Section of growth after introduction of radon seeds consists mainly of fibrous tissue and epithelial debris. Some cell proliferation present, probably chronic inflammatory. No evidence of original typical carcinomatous picture. ($\times 65$.)

AUTOPSY.—A fistula led to the anterior surface of the duodenum. It was difficult to identify the growth, the remains of which showed several necrotic areas. A portion was removed for histological examination, and many sections were cut, one of which is illustrated in *Fig. 45*. None of the sections showed cancer cells to be present. No secondary deposits were found. The wall of the duodenum was intact at the site of the growth.

SUMMARY.

1. Cancer of the duodenum is an extremely rare condition, but the duodenum is the commonest site of small-intestinal cancer. The ampullary region is most often affected.

2. It has been shown that while the duodenum is sometimes microscopically involved in pyloric cancer, it possesses considerable resistance to its spread.

3. Duodenal ulcer has not been clearly proved to be a predisposing cause of duodenal cancer.

4. Cases of obstructive jaundice should be submitted to laparotomy with the exceptions mentioned above.

5. In the case reported, had radium been available at the first operation, the chances of the patient would have been enhanced considerably, since a second incision into the duodenum would have been avoided. The introduction of radon seeds around the growth adds very little to the time of a palliative operation, and since metastases occur only late on in this condition, this treatment would appear preferable to the more radical measures already discussed, and to offer a greater hope of a permanent cure.

In conclusion I wish to thank Dr. Effie Brander for the preparation and description of the histological specimens.

REFERENCES.

- ¹ SCHLESINGER, *Wien. klin. Woch.*, 1898, x, 245.
- ² PERRY and SHAW, *Guy's Hosp. Rep.*, 1893, i, 274.
- ³ FENWICK, W. S., *Edin. Med. Jour.*, 1901, x, 309.
- ⁴ MAYDL, quoted by Notlingel.
- ⁵ NOTLINGEL, *Diseases of the Intestines and Peritoneum*, American ed., 1905.
- ⁶ NAGEL, G. W., *Arch. of Surg.*, 1925, ii, 529.
- ⁷ EUSTERMANN, G. B., BERKMAN, D. M., and SWAN, T. S., *Ann. of Surg.*, 1925, lxxxii, 153.
- ⁸ KOHLER, quoted by Jefferson.
- ⁹ DEAVEH, J. B., and RAVADIN, I. S., *Amer. Jour. Med. Sci.*, 1920, clix, 469.
- ¹⁰ JEFFERSON, *Brit. Jour. Surg.*, 1916-17, iv, 209.
- ¹¹ BLAND-SUTTON, SIR JOHN, *Trans. Med. Soc. Lond.*, 1914-15, xxxviii.
- ¹² ROLLESTON, SIR H. D., *Lancet*, 1901, i, 1121.
- ¹³ GEISER, J. F., *Deut. Zeits. f. Chir.*, 1907, lxxxvi, 41.
- ¹⁴ ORTH, *Lehrb. spec. Path.*, 1887, i, 850.
- ¹⁵ WALTON, A. J., *Nelson's Loose-Leaf Living Surgery*, v, 95.
- ¹⁶ LETULLE, *Bull. Soc. Anat.*, 1897, lxxii, 721.
- ¹⁷ DEWES, J. W., and MORSE, G. W., *New Eng. Jour. Med.*, 1928, cxviii, 383.
- ¹⁸ COLLIN, *Thèse de Paris*, 1894.
- ¹⁹ EWING, *Neoplastic Diseases*, 1928, Philadelphia.
- ²⁰ OUTERBRIDGE, G. W., *Ann. of Surg.*, 1913, lvii, 402.
- ²¹ COHEN, I., and COLP, R., *Surg. Gynecol. and Obst.*, 1927, xlv, 332.
- ²² PAUCHET and LUQUET, *Bull. de l'Acad. de Méd.*, 1927, xcvi, 276.

TUBULAR HERMAPHRODITE WITH TERATOMA OF THE INTERNAL GENITALIA.

By E. D'ARCY McCREA.

ASSISTANT SURGEON AT SALFORD ROYAL HOSPITAL, MANCHESTER.

THE patient, age 31 years, was admitted to the male wards of the Salford Royal Hospital in May, 1928; he complained of severe pain in the back, obstinate constipation, and a swelling of the abdomen which was rapidly increasing in size.

ON EXAMINATION.—A large abdominal tumour was obvious, apparently arising from the pelvis; it was firm, and in size corresponded approximately to that of a 6-to-7-months pregnancy; per rectum a mass could be felt anteriorly. Neither testis was present in the scrotum, but the patient stated that at times both had appeared.

OPERATION.—Laparotomy revealed a large nodular tumour, in parts cystic, in others solid, emerging from the pelvis, and to the posterior aspect of which the rectosigmoid was adherent. It was considered to be a malignant tumour, probably arising from a retained testis, and inoperable. However, on searching for what were assumed to be the testes, both were found; but it was noted with some surprise that a tube with fimbriated extremity lay in relation to each. The size of the tumour prevented a thorough exploration of the pelvis, and the abdomen was closed.

POST-MORTEM EXAMINATION.—The patient died in July, 1928, and at the autopsy the peritoneum was studded with secondaries, there was a mass of enlarged aortic glands, and the liver also showed numerous secondaries; there were no metastases elsewhere. The tumour and pelvic contents were removed *en masse*, and on dissection I found the following interesting condition (*Fig. 46*). There were present a uterus with broad ligament and tubes with fimbriated extremities passing to the two gonads previously noted; two vasa deferentia passing down the postero-lateral margins of the uterus in intimate relation with its wall; two seminal vesicles lying behind and to each side of the lower segment of the uterus; and a well-developed prostate. The uterine cavity, which showed no evidence of division into uterus and vagina except for a narrowing $2\frac{1}{2}$ cm. from its inferior termination, opened into the prostatic urethra at the normal site of the utriculus prostaticus, and the length of its cavity from urethra to apex was 13.25 cm. The vasa deferentia took origin from two nodules lying between the layers of the broad ligament, and situated medial to the two gonads, but clearly separate from them; they joined the ducts of the seminal vesicles, and the ejaculatory ducts opened at their normal sites on either side of the utriculus.

The tumour ($20 \times 13 \times 7$ cm.) appeared to be a teratoma, and had an attachment to the antero-superior surface of the uterus and to the anterior

aspect of the broad ligament. It had no relation to the two gonads or to the tubes, all of which were free and clearly demonstrated at the operation

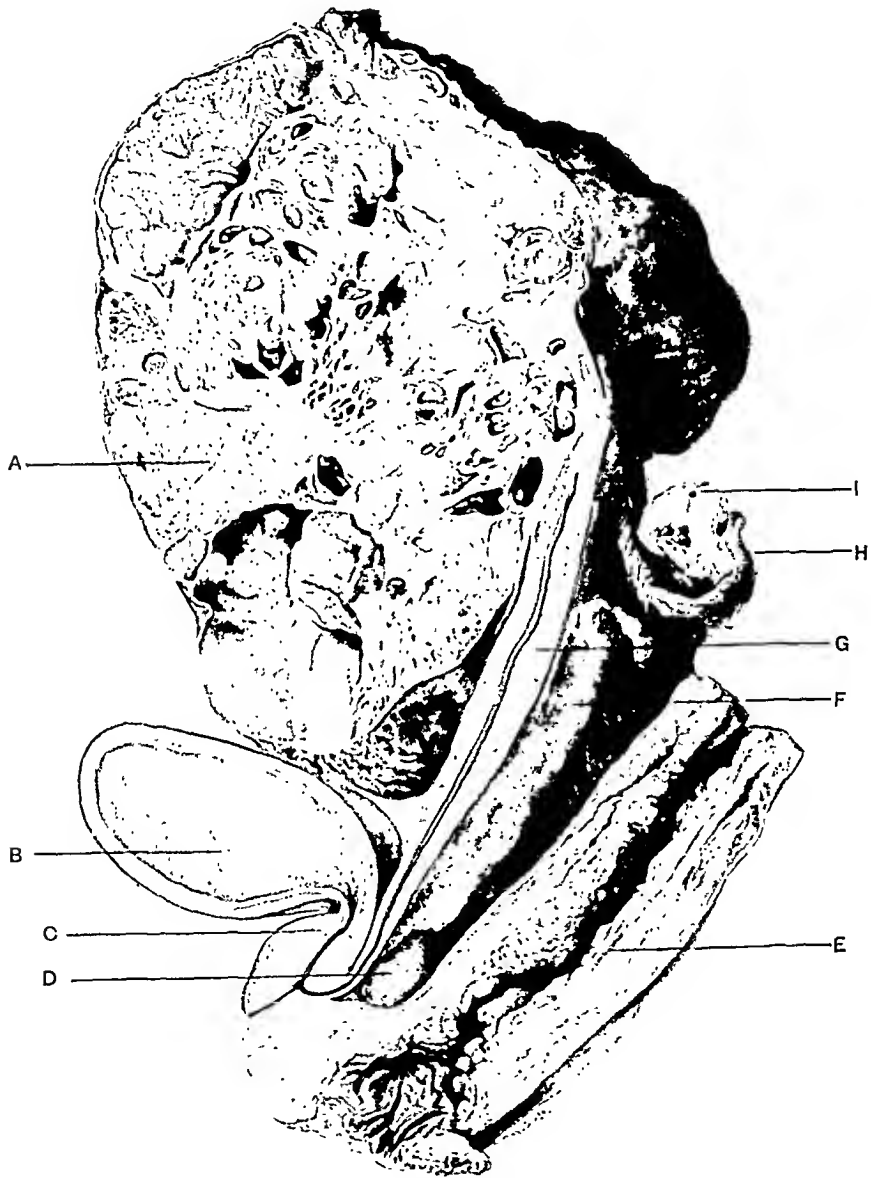


FIG. 46.—Sagittal section through tumour, genito-urinary organs, and rectum. A, Teratoma; B, Bladder; C, Prostate; D, Seminal vesicle; E, Rectum; F, Vas deferens; G, Uterus; H, Fallopian tube; I, Testis.

in May, although post mortem the appendages of the left side were partially obscured by the tumour and its secondaries.

MICROSCOPICAL EXAMINATION.—The following facts were revealed: The uterus possessed an apparently normal endometrium and a wall made up of smooth muscle; the tubes, seminal vesicles, vasa deferentia, and prostate

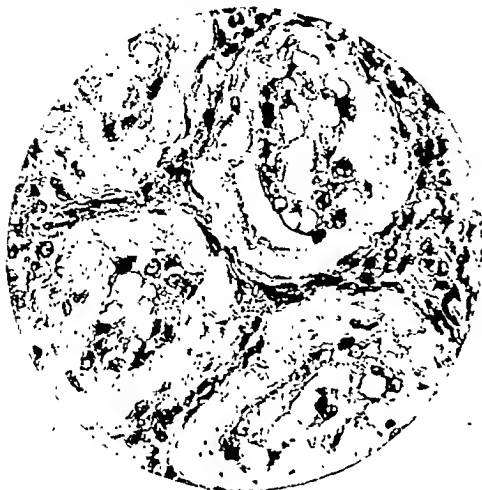


FIG. 47.—Microphotograph of the right gonad (high power).

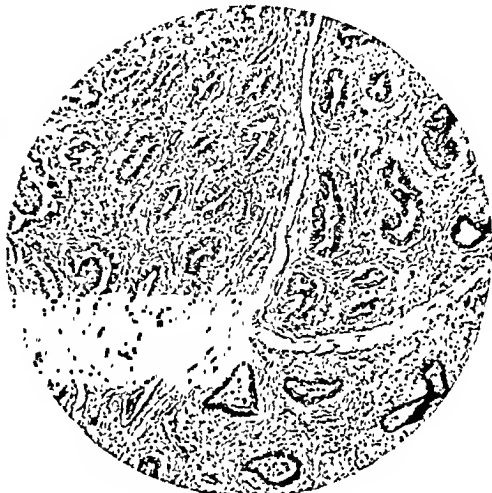


FIG. 48.—Microphotograph of the left gonad (low power).

were of normal appearance. The gonad of the right side was quite definitely a testis (*Fig. 47*), and serial sections failed to show any trace of ovarian tissue; the left gonad, which was largely destroyed by secondaries, also proved to be a testis (*Fig. 48*). The tumour contained numerous cysts, and the majority of these showed intracystic papillomata; amongst other tissues found were bone, cartilage, smooth muscle, and epithelial-lined tubules. Ectodermal derivatives identified were some rudimentary teeth and a small cyst lined by squamous epithelium from which a few hairs sprang. All the secondaries examined were of the nature of papilliferous cystadenomata (*Fig. 49*).



FIG. 49.—Microphotograph of a portion of the tumour showing the structure of a papilliferous cystadenoma. All the secondaries were of this nature.

In appearance this patient was typically masculine, tall, lean, and muscular, with broad shoulders and narrow hips, a heavy growth of beard, and a deep voice. The pubic hair was distributed after the male fashion, and the penis was of more than

average size; he appeared to be a double cryptorchid, and the scrotum though small was perfectly formed. There was no abnormal development of the breasts.

Such history as was obtained from the patient was meagre; he stated that the tumour had been present for six months. Subsequently the following history was obtained from two brothers, one of whom had shared the patient's room since 1919. An apparently normal interest in the female sex had been exhibited and he was engaged to be married; he had never taken part in games and was acutely self-conscious of the absence of the testes. Since about the age of 17 there had been complaints of periodical attacks of abdominal pain, "nearly every month, periodical like a woman" as one brother volunteered; at times it had been observed that the urine in his chamber was blood-stained and contained "strings of blood". No history of seminal emissions or of coitus could be obtained. His brothers suspected that the tumour had been present for years.

The question of true hermaphroditism has often been both fully and bitterly debated, and is reviewed at length by Lacassagne;¹ a recent case published by Masson² is of interest in relation to this article. Whether living gametes of both sexes must be produced, or whether mere 'potential' or 'accidental' hermaphroditism should be admitted as true, appears a profitless discussion. Klebs³ classification of true and false hermaphroditism, and his subdivisions of these, are sufficiently widely accepted to be utilized, and the case described above comes under his heading of false hermaphroditism. For descriptive purposes the classification developed by Guéricolas⁴ into glandular and tubular hermaphroditism is most useful, the latter coming under the heading of false hermaphroditism. My case is therefore one of tubular hermaphroditism—that is, of persistence of both the Müllerian and Wolffian ducts in the one being. Such examples are rarities, and the recorded instances may be discovered in the writings of Simpson,⁵ Primrose,⁶ and of Greig and Herzfeld.⁷

The occurrence of a neoplasm arising from the internal genitalia of these hermaphrodites is a greater rarity, and the number recorded in the literature is small; Neumann⁸ has collected and discussed such cases. He separates them into two groups: (1) The solid large-celled carcinomata; and (2) The adenomata tubularia testis, the latter group being a very definite entity. Of the former, with which we are more particularly concerned, he has found 27 instances in a search of the literature, 25 in pseudo-hermaphrodites and 2 in true hermaphrodites. Some of these have been examples of that peculiar tumour, epithelioma chorio-ectodermale, and undoubtedly of teratomatous origin; others have been variously described as seminoma, sarcoma, and carcinoma, and many writers would now ascribe to these a teratomatous origin. Pick⁹ has discussed the relation of adenoma tubulare testis to teratomatous formations.

Since the publication of Neumann's paper two other cases which may be placed in his first group of the large-celled carcinomata have been recorded by Pettavel¹⁰ and by Christophe, Firket, and Hogge.¹¹ Another instance which is not included in his list is that of Marchand.¹²

*Case 1 (Pettavel).—*Male external genitalia with peno-serotal hypospadias, prostate present, left testis palpated in the scrotum, but the right absent. A history of seminal emissions was given. Laparotomy revealed an atrophic uterus, a right Fallopian tube, the left being absent, and a right-sided tumour. Diagnosis: ovarian carcinoma.

*Case 2 (Christophe).—*Male, age 25 years. Until 18 years of age educated as a girl. On examination general aspect male, the penis was small with vulviform hypospadias, the scrotum poorly developed, and the testes absent. Rectal examination failed to reveal a prostate or seminal vesicles. A history of seminal emissions was elicited. On laparotomy there were found an atrophic uterus and vagina, and a left Fallopian tube and gonad, but on the right side the cornu of the uterus was continuous with a ligament which became attached to a tumour the size of a fist. The left gonad proved to be an ovitestic in relation both to a Fallopian tube and to an epididymis and vas deferens: together with the tumour on the right side was a smaller ovitestic. Diagnosis: seminoma.

*Case 3 (Marchand).—*Male, age 24 years, a double cryptorchid. A uterus and vagina, which opened into the prostatic utricle, the prostate, both seminal vesicles, and the left vas deferens were present, but the right vas was absent. A left undescended testis was found, but the right gonad was replaced by a tumour; secondaries were present in the liver and abdominal lymphatic glands. Diagnosis: large round-celled carcinoma.

In its histology the tumour described above belongs to the atypical teratoma, and differs from the thirty previously noted; furthermore, like that of Christophe, it did not appear to take origin from a gonad, as is usual with these. However, it seems possible that its origin was from sex cells, and, although ovarian tissue was not discovered, it may be that the patient was a glandular or true hermaphrodite and the tumour took the place of a destroyed ovary. Some colour is lent to such a view by the nature and predominance of the proliferating papillary cysts, which were most reminiscent of ovarian papilliferous cystadenomata, as also by the history obtained, which in several particulars suggested ovarian activity.

SUMMARY.

An example is recorded of a tubular hermaphrodite in whom an atypical teratoma of the internal genitalia had developed.

REFERENCES.

- ¹ LACASSAGNE, *Gynéc. et Obst.*, 1920, i, 273.
- ² MASSON, *Amer. Jour. Obst. and Gynecol.*, 1925, ix, 81.
- ³ KLEBS, *Handbuch d. pathol. Anat.*, 1873, Berlin.
- ⁴ GUÉRICOLAS, *Thèse Lyon*, 1899 (quoted by Lacassagne).
- ⁵ SIMPSON, *Works of Sir J. Y. Simpson*, 1871, ii, 463, Edinburgh.
- ⁶ PRIMROSE, *Jour. Anat. and Physiol.*, 1898-9, xxxiii, 64.
- ⁷ GREIG and HERZFELD, *Edin. Med. Jour.*, 1927, xxxiv, 701.
- ⁸ NEUMANN, *Arch. f. Gynäkol.*, 1927-8, cxxxi, 477.
- ⁹ PICK, *Arch. f. mikroskop. Anat.*, 1914, lxxxiv, 119.
- ¹⁰ PETTAVEL, *Schweiz. med. Woch.*, 1926, lvi, 447.
- ¹¹ CHRISTOPHE, FIKET, and HOGGE, *Ann. d'Anat. pathol.*, 1927, iv, 989.
- ¹² MARCHAND, *Berl. klin. Woch.*, 1900, xxxvii, 467.

RODENT ULCER AS THE SEQUEL TO PROLONGED X-RADIATION.

BY HAROLD EDWARDS, E. FF. CREED, AND H. A. COLWELL,
KING'S COLLEGE HOSPITAL, LONDON.

THE patient who is the subject of the present note had a radical amputation of the left breast for carcinoma in October, 1913. Healing was uneventful, and a healthy scar resulted. In July, 1914, she commenced a series of X-ray treatments as a prophylactic against recurrence, and it is to the large number of these that attention is specially directed. The patient was, on her own statement, very worried about the possibility of recurrence, and pressed for continued and repeated treatment. The treatments were given at varying intervals, and may be summarized as follows:—

	July to December	1914	1915	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925
Number of treatments }		9	16	14	10	10	8	7	4	4	4	4	4

In all 94 treatments were given. The filters used varied; for the first ten treatments a filter of 1 mm. of aluminium was employed; the following thirteen were given through aluminium filters of 2 mm. in thickness, and in the remainder, the filter, again of aluminium, was 3 mm. in thickness. The dosage was estimated by the Sabouraud pastille, and in each case one full pastille dose was given, the pastille being placed on the side of the filter remote from the tube, i.e., towards the patient's skin.

In August, 1927, the patient came to one of us (H. A. C.), who had not seen her before, for inspection, and with an urgent request that X-ray treatment might be continued. The impossibility of this was pointed out when inquiries had been set on foot regarding past X-ray treatments. Examination of the patient at this time showed a healthy scar, but the skin in the neighbourhood was atrophic and was covered with telangiectases. The skin of the right breast was also affected in a similar way, but not to so marked a degree. From time to time the patient came up for inspection, and in December, 1928, wrote to say that she had a small sore at the upper end of the operation scar. It was found to be covered with a dry scab, which the patient was advised to remove by fomentations. She was not again seen until February, 1929, when the ulcerated area was found to have increased in size.

The ulcer, which interrupted the line of the scar on the chest wall near its upper end, was roughly circular in outline. The edge of the ulcer was firm, and slightly raised, and the base was covered with a sanious coagulum. The clinical appearances were those of a rodent ulcer. Two minute nodules could be felt in the skin just to the medial aspect of the ulcer, and the skin

around was very atrophic and covered with telangiectases (*Fig. 50*). A biopsy confirmed the clinical diagnosis of rodent ulcer, and removal of the ulcer was advised.

The ulcer, including a wide area of skin, was removed under gas and oxygen anæsthesia. Despite wide undercuttering of the skin, it was found impossible to obtain close apposition of the edges of the wound, and a

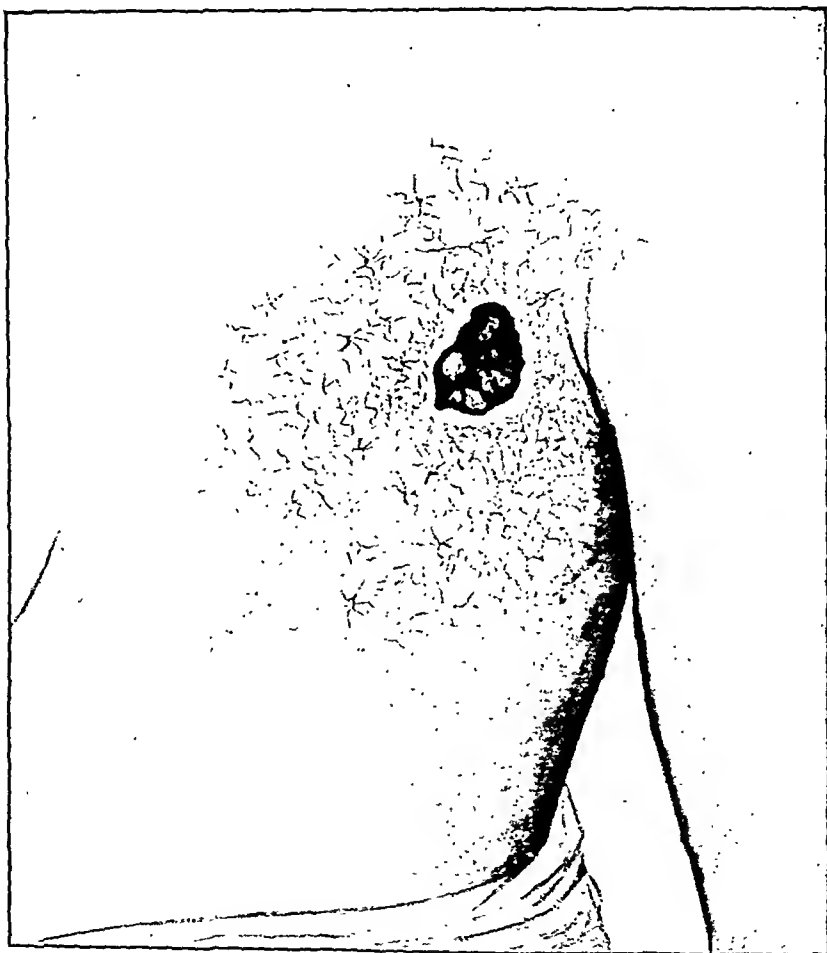


FIG. 50.—The condition when first seen. Note the telangiectases surrounding the ulcer.

triangular area of subcutaneous tissue was left uncovered. A skin-graft was applied to this area ten days later.

The whole of the tissue was embedded for section cutting, and microscopic examination of a whole section was made. The microscopical appearances were as follows :—

The section shows a piece of tissue covered, except for an ulcerated area

about a quarter of an inch wide, by a keratin-forming stratified epithelium. The epithelium is thin, but shows some hyperkeratosis and dyskeratosis. There is almost complete absence of papillae. There is some cellular infiltration in places, immediately beneath the epithelium, with lymphocytes and plasma cells. There is much hyaline degeneration of the corium. In the central part there is an ulcerated area beneath which are masses of cells showing the characteristic features of a basal-celled carcinoma. These cells are arranged chiefly in large blocks with a peripheral palisade layer of columnar cells (*Fig. 51*). There is some mitosis present. In some areas the cells tend to assume a spindle form, but there is no tendency to production of prickle cells

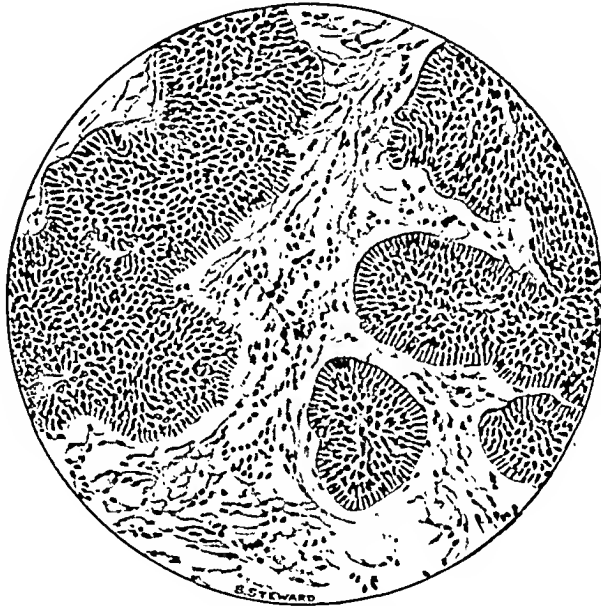


FIG. 51. —Microscopical appearance of ulcer. ($\times 160$). For description, see text.

or keratin. In some areas, particularly towards the centre of the lesion, the carcinoma shows a more reticular arrangement with areas of cystic degeneration. The carcinoma has invaded beneath the surrounding epithelium, but remains very superficial.

Comment.—It is perfectly clear from the pathological findings that the ulcer was not a recurrence of the primary growth. It is difficult to resist the view that the ulcer was a sequel to the large amount of radiation which had been given, and if so, the case is of interest from the extreme rarity of the condition. Squamous-cell carcinomata are of course well known as sequelæ to excessive radiation; but rodent ulcer is excessively rare, and indeed, in our experience, this case is unique.

VOLVULUS OF THE STOMACH.

BY JAMES BUCHANAN.

RESIDENT SURGICAL OFFICER, ST. JAMES' HOSPITAL, LONDON.

A REVIEW of the literature concerning this remarkable condition shows that it is of sufficient rarity to merit comment. The following case history demonstrates a number of the accepted etiological factors and clinical signs, and has the additional advantage that on post-mortem examination the exact condition present was reproduced, and the morbid anatomy studied rather more carefully than is possible in the operating theatre when dealing with an extremely collapsed patient.

CASE HISTORY.

A. W., female, age 74, unmarried, was admitted to St. James's Hospital at 11.30 p.m. on Dec. 26, 1929, with a provisional diagnosis of gall-bladder disease.

HISTORY.—The woman had lived in a single room without companions, and little is known of her previous history. Relations, whom she visited early in December, however, state that she was exceptionally healthy, and complained of no pains or discomfort when she was last seen. With regard to the present condition, she had not been feeling very well for some days, but made a hearty dinner, which included plum pudding, on Christmas Day. In the night she was seized with pain in the upper abdomen and retching, which continued up to the time of admission.

ON ADMISSION.—The patient complained of pain round the umbilicus and in the back—duration thirty-six hours.

ON EXAMINATION.—She was a thin elderly woman of the viscerotonic type, pale and anxious, and with a tendency to retching at intervals. Temperature 97.8°, pulse 84, respiration 36. Tongue dry; breath not offensive.

Abdominal examination showed a narrow subcostal angle, and there was some generalized distension of the upper abdomen. No peristaltic waves were noted. There was tenderness all over the upper abdomen, but it was difficult to discover any definite tender point, as any pressure tended to cause an attack of retching. The lower abdominal quadrants appeared normal, and nothing abnormal was noted on pelvic examination.

The heart-sounds were faint but clear, and the apex beat was not displaced. There appeared to be diminution of air entry at the bases of the lungs, and fine crepitations were present at each base. Provisionally the diagnosis of pyloric stenosis was made. The patient was given a hypodermic injection of morphia $\frac{1}{2}$ gr., atropin $\frac{1}{100}$ gr. She dozed through the night and had several attacks of retching after attempting to take drinks of water, the fluid being returned practically unchanged. No actual vomiting of stomach contents occurred while she was under observation.

Next morning a swelling was obvious under the umbilicus reaching to the false pelvis, but separable therefrom. There was less distension of the upper abdomen. The swelling had the peenliar characteristic of being dull in places and resonant in others. Laparotomy was undertaken at once.

OPERATION.—Anæsthesia was induced with open ether, and a right paramedian incision was made with its centre rather below the umbilicus. As soon as the peritoneum was opened, a small quantity of blood-stained fluid escaped and a swelling

covered with a thin layer of omentum presented. Investigation of the relations of this swelling showed that it had the transverse colon above it, that it was free from the pelvis but extended upwards, and that it was bilocular. It was impossible to separate the thin layer of omentum from it without incision. An opening was

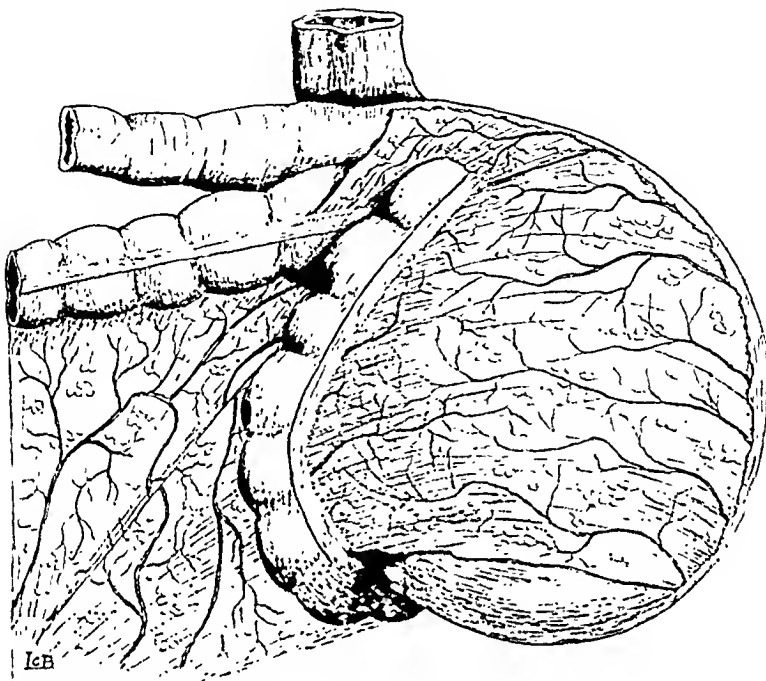


FIG. 52.—Post-mortem appearance of stomach in mesenterio-axial volvulus. (Scmidgrammatic.)

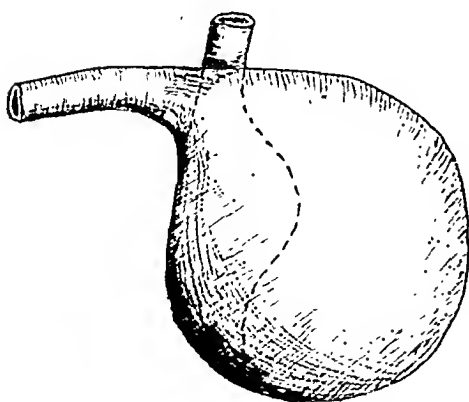


FIG. 53.—Mesenterio-axial volvulus.

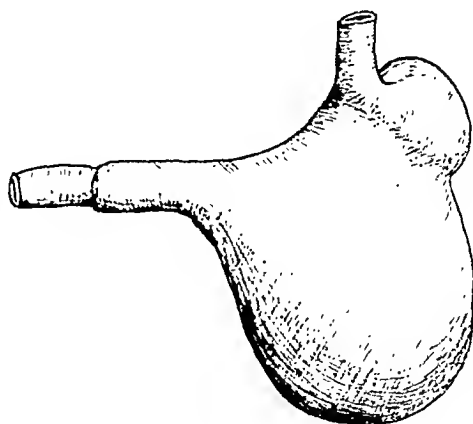


FIG. 54.—Stomach after reduction.

therefore made through the omental covering of the cystic swelling, and a bluish wall presented. Towards the lower end of this, large tortuous veins were seen passing into the omentum at its attachment there, and thus the greater curvature of the stomach was recognized.

The abdominal incision was enlarged upwards and the entire stomach delivered from the wound. It was then seen to be in a state of 180° volvulus. This was reduced without tapping and the distended stomach in its now normal relationship was returned to the abdominal cavity. A stomach tube was then passed by the anaesthetist and guided through the cardia. A large quantity of gas escaped and then 44 oz. of offensive blood-stained material were withdrawn. Considerable improvement in the general condition was now noted.

Nothing further was attempted. The abdominal wound was closed in layers and the stomach tube retained for the next four hours to prevent any immediate gaseous re-distension. Saline and glucose solution was administered by continuous proctoclysis and a radiant-heat bath applied. Unfortunately, however, the general condition again deteriorated, the pulse weakened, and the patient died some six hours after the operation.

POST-MORTEM EXAMINATION (in collaboration with Dr. A. P. Piggot).—The reduction of the volvulus had persisted, and the stomach appeared as in *Fig. 54*. The pyloric canal was $3\frac{1}{2}$ in. long, narrow, and extremely mobile. The gastro-hepatic omentum was stretched longitudinally, the result of habitual ptosis.

The stomach maintained its bilocular form, there being a line of congestion running vertically across the anterior surface between the cardiac third and pyloric two-thirds, giving the appearance of a congenital diverticulum in the fundus. This line gave the axis of the volvulus. The layers of the greater omentum had been accurately and completely separated, so that the lesser sac extended right into the apex of the omentum.

The pyloric mobility was such that it was an easy matter to reproduce the volvulus found at operation. The axis of twist was at right angles to the curvatures. The pyloric antrum passed forwards and to the left, and also rotated so that the posterior surface of the stomach came to look forwards. The cardiac end had passed slightly backwards, and to the line of constriction then formed the bilocular appearance was due. The posterior surface, looking forward, had been largely responsible for the opening out of the omental layers.

As the pyloric antrum passed across the abdomen, the colon had been drawn with it, and thus apparently the colon had lain in front of the stomach. In reality there was a loop of colon lying in front of the volvulus, the remainder of the transverse colon then passing onwards behind and below the distended stomach. The large veins noted during operation were situated on the posterior aspect of the greater curvature.

The stomach was then opened. A quantity of currants were found remaining in the stomach, the remains, presumably, of the meal taken on Christmas Day. There was diffuse hæmorrhagic infiltration of the mucous membrane, but there were no signs of necrosis or gangrene. The line of congestion noted on the peritoneal surface was not noticeable on the mucous surface. Very careful scrutiny revealed no trace of any ulcer or ulcer scar, and there was no evidence to suggest that primarily there had been an hour-glass stomach.

MORBID ANATOMY.

Volvulus of the stomach may be *total* or *partial*. It must be pointed out that the total form is not strictly a complete volvulus from the anatomical point of view, the gastro-phrenic ligament being sufficiently strong to hold the left side of the fundus steady even in extreme torsion. A total volvulus implies that the entire organ has taken part in a twist of 180° round a definite axis; a partial volvulus that only a portion of the viscus is concerned.

Total Volvulus.—The cases recorded may be divided into two groups, according to the axis of rotation, as first suggested by von Haberer. We may therefore recognize two main types: (1) Volvulus mesenterio-axialis (*Fig. 55*); (2) Volvulus organo-axialis (*Fig. 56*).

Volvulus Mesenterio-axialis.—The majority of recorded cases are of the mesenterio-axialis type. In this form the rotation is from right to left around a vertical axis at right angles to the cardiopyloric line. The extent is rather less than 180° and the main movement is effected by the pyloric end of the stomach, which is unduly mobile. The pyloric end always passes in front

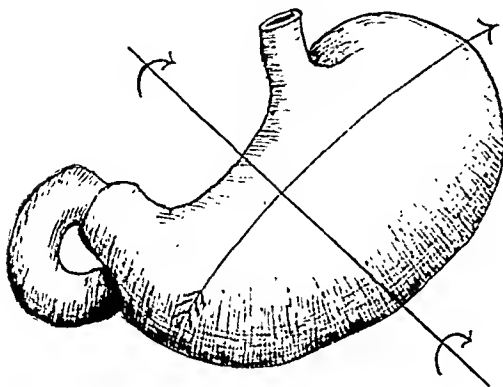


FIG. 55.—Volvulus mesenterio-axialis. (After Kocher.)

of the fundal end from the right below, upwards, and to the left; a small reciprocal movement is often seen at the fundus. The anterior wall, sharply kinked on itself, now looks backwards and to the left, while the posterior wall presents under the abdominal wall, veiled, in the majority of cases, by the great omentum.

Displacement of organs in this type is not extreme. The colon, particularly its right half, is carried by the pyloric end of the stomach through its omental attachment upwards and in front of the vol-

vulus, there forming a loop, so that there is a rising limb, a sharp kink, and then a descending limb which passes below and behind the volvulus to appear on the left again, rising towards the splenic flexure. The remaining abdominal viscera are not grossly displaced as a rule.

Kocher described a case of this type, the interesting point being that it was a total volvulus occurring in an hour-glass stomach. The other cases of volvulus in hour-glass stomach have been of the partial type.

Volvulus Organo-axialis.—This type of volvulus is seen when the axis of rotation is the line joining the cardiac and pyloric orifices. Two subgroups are noted here, in the first of which—organo-axialis posterior—the stomach twists from left to right and from below upwards. The extent of the rotation is about 180° , and when complete the anterior surface looks backwards while the posterior surface now presents under the abdominal wall, veiled by the mesocolon when the colon has participated in the upward twist, as is usual. In some undoubted cases the colon has remained below, and so two varieties of this group may be differentiated—*infracolic* and *supracolic*. The *supracolic* variety presupposes a long lax great omentum or actual rupture of this mesentery (Kerr).

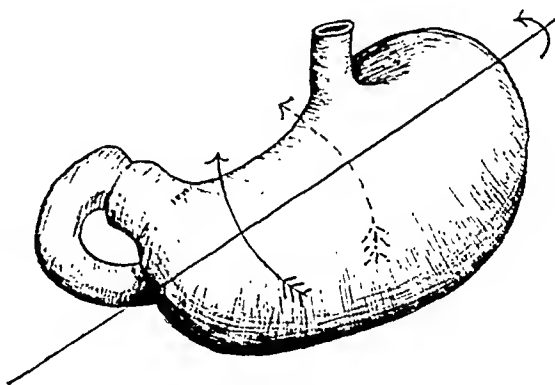


FIG. 56.—Volvulus organo-axialis. (After Kocher.)

There is displacement of neighbouring viscera, particularly the colon, the spleen, and the pancreas. The latter viscera are easily replaced when the volvulus has been reduced; but the colon, wedged between the diaphragm and the distending stomach, undergoes distension in its right half and usually offers a formidable barrier to replacement, unless the stomach is first emptied by tapping with a trocar. The degree of twist appears to be variable. The twist is usually about 180° . Thorek, Pendl, and Kocher mention 270° and Niosi 360° , but it would appear that figures estimated by surgeons operating rapidly should be accepted with reserve. Berti, however, described a post-mortem case, and his description permits of no doubt.

Two cases have been recorded of the subgroup organo-axialis anterior. They are difficult of interpretation. Neumann and Delangre described cases in which, though the axis was primarily the same, the sense of rotation was changed. The greater curvature passes backwards and then upwards around the cardiopyloric axis, and the summit of the volvulus appears in the lesser sac, its posterior wall raising the lesser omentum. These two surgeons found a large fluid tumour, divided into two parts by a furrow at the lesser curvature. The posterior wall was covered by the stretched gastro-hepatic omentum, while the anterior gastric wall lay above and behind, uncovered by any omentum. From experimental evidence Tuffier and Jeanne found it difficult to accept Neumann's statement that there was rotation of 270° in his case. They conclude that rotation of this type cannot exceed 180° , and that this is scarcely a regular form, as the anterior wall is much less displaced than the more dependent portion of the stomach at the greater curvature.

Total volvulus has also been recorded in association with intrinsic gastric lesions. It is necessary that such lesions shall in no way impede the free mobility of the stomach as a whole; the lesion must therefore be situated near the cardiac orifice. Berg mentions a volvulus associated with carcinoma of the cardia, while Sinjuschin found an ulcer near the opening of the œsophagus. These lesions probably had little or nothing to do with development of volvulus in these cases.

Partial Volvulus.—Volvulus organo-axialis anterior is not a true total volvulus, for displacement of the moving parts is so unequal. The greater curvature passes through 180° while the anterior face is displaced only a quarter turn or less. Thus it is a limited volvulus. In a number of observations the cardiac portion of the stomach remains in place, while the pyloric portion rotates from behind to the front and from below upwards. There are then two parts to be seen, an upper vertical portion on the left, and another below, horizontal, and twisted on an axis. The pylorus becomes kinked and occluded, and distension occurs in the pyloric pouch, but never to the extent seen in total volvuli. This type is always associated with gastric or extragastric lesions. In some cases a benign gastric tumour appeared to be the cause. Payer mentions three such examples. At other times perigastritis fixes one half of the greater curvature and allows the remainder to twist into a partial volvulus.

The commonest lesion found, however, is the hour-glass stomach. A pyloric pouch is produced, able to turn on its extremities like pivots, the stenosed area on the left, the pylorus on the right. There is no ligamentous

fixation for the pouch so formed. There may be, however, some adhesions to the pancreas and perigastritis, which maintain the stenosis and give a fixed point round which rotation may more readily occur. The twist in these cases is always less than with total volvuli, distension is less marked, and there is no marked interference with the blood-supply at any stage.

Some partial volvuli have been associated with congenital malformations such as diaphragmatic herniæ, tears in the transverse mesocolon (Hermes), or tears in the gastro-hepatic omentum (Niosi, Gill). Payer, in 500 cases of diaphragmatic herniæ, found 12 instances of partial volvulus.

PATHOLOGY.

The following classes of volvuli may be recognized: (1) Associated with diaphragmatic herniæ or congenital openings in the mesenteries; (2) With intrinsic gastric lesions, e.g., perigastritis, gastric ulcers, tumours; (3) Idiopathic. Cases in the first two classes are usually of the partial variety. In the latter case volvulus is usually total and acute.

Considering the extent of the twist in many cases, there is remarkably little interference with the circulation. Congestion is the rule, but the blood-vessels are never obstructed sufficiently to cause early necrosis or gangrene. In late neglected cases the vessels may become occluded by the extreme distension, and should the patient survive to that stage, final rupture of the stomach may occur. It has been suggested that a number of the cases of 'idiopathic' rupture of the stomach found at post-mortem examination are in reality late unrecognized cases of total volvulus.

Partial volvulus, being in general subacute and having only minor interference with the vessels at any stage, never reaches the stage of rupture. In incompletely obstructed cases marked gastric hypertrophy may result. In cases of volvulus of any standing, the mucous membrane is usually mottled with small submucous hæmorrhages and the contents are gas and blood-stained fluid containing food debris. The peritoneum in these cases usually contains some free serous fluid, which is often blood-stained.

It has been suggested that the distension, which is so marked a feature, may in some cases be a primary condition, i.e., that acute dilatation occurring in a markedly ptosed stomach may be the actual cause of the volvulus. Once volvulus has commenced, distension is marked and progressive. Experimentally it has been shown that up to a certain degree of torsion (less than 180°), excessive distension actually tends to reduce or diminish the extent of the rotation. Once the stomach has become incarcerated, however, distension only serves to maintain its abnormal position.

It has also been shown by experiment (Tuslier) that one of the first effects of rotation is to close the pylorus by combined kinking and twisting. The pylorus approaches the cardia and a deep V appears in the lesser curvature in both organo-axial and mesenterio-axial types. This midgastric constriction increases as the distension increases, and in advanced organo-axial cases the pylorus may be approximated to the cardia. Distension progresses more rapidly once the cardiac orifice has been occluded, and may be extreme.

In some cases there is marked distension in the transverse colon as well. This is particularly noted in the organo-axial type. The colon is caught

between the upturned stomach and the dome of the diaphragm. Marked distension in the right half of the transverse colon then occurs. It may be so marked as to resist any attempt at reduction at operation, and this is one of the reasons why tapping by trocar is so often necessary before the volvulus can be reduced.

Various complications dependent upon the volvulus of the stomach have been described. Borchardt had to perform splenectomy in his case for rupture of the splenic vessels and profuse hæmorrhage. Wiesinger and Hailes noted some patches of fat necrosis in the vicinity of the tail of the pancreas. Both these cases recovered without ill effects. Rupture of the mesenteries has also been noted, particularly by Kerr, who found that the greater omentum had been completely torn away from the greater curvature.

ETIOLOGY.

The condition under consideration is one of extreme rarity, the majority of recorded cases being mentioned in this paper.

The sex incidence is equal, and most of the cases occurred in persons past middle age, though three cases (Dujon, Krynholtz, Siegel) have been recorded in the very young.

Analysis of the supposed predisposing causes reveals that in the majority of cases which developed acute total volvulus there was no history of preceding gastric symptoms, the patients usually being in good health; but in every case stress is laid on the degree of *ptosis* present in the reduced organ. The laxity of the ligaments, the result of the habitual ptosis, allowed the development of a potential space beneath the diaphragm for the accommodation of the viscus, once the twist had occurred. On the other hand, the cases of partial volvulus almost always had had symptoms suggesting a gastric lesion. Perigastritis, gastric ulcer, and hour-glass stomach, particularly the latter, have all been described as possible predisposing causes in this type.

French writers lay particular stress upon 'aërocolie' as an exciting cause, the theory being that the gas-distended colon tends to lift the body of the stomach forwards and upwards and so produce a position more favourable for volvulus, should other factors be favourable. In cases where volvulus is associated with internal hernia, the presence of the congenital opening in the parietes is without doubt a predisposing factor.

The immediate exciting causes are over-filling of the stomach, vigorous peristalsis rapidly followed by an antiperistaltic wave, and, possibly, mechanical torsion of the ptosed organ, as in vomiting. Pendl suggests that a thick gastrocolic omentum together with strong colonic peristalsis, by dragging the greater curvature up towards the left dome of the diaphragm, produced volvulus in his case. Kerr suggests acute dilatation in a ptosed stomach from 'loss of splanchnic control'. There is apparently no doubt that two cases at least were due to trauma of the abdominal wall.

In the organo-axialis type it would appear that the actual volvulus is produced by an exaggeration of the normal forward movement of the left border of the stomach in peristalsis (as seen by X rays), the result of extremely vigorous peristaltic waves. For the mesenterio-axialis type excessive peristaltic waves in a long over-loaded loop with a narrow basis of attachment

is clearly the mechanism. It is exactly comparable with the commoner volvulus of the sigmoid colon, where the same factors come into play.

Partial volvuli, especially those associated with hour-glass stomach when the pyloric pouch turns upwards, also appear to be due to abnormal peristalsis. The importance of 'aërocolic' lifting the pouch upwards is particularly impressed in this type.

SYMPTOMATOLOGY.

Total Volvulus.—There are usually no premonitory symptoms. The onset is characteristically sudden and the symptoms are progressive in nature. Epigastric pain, often very severe, and vomiting are the earliest manifestations. Severe pain in the back has been noted in a number of cases. The vomitus consists of stomach contents only, and soon ceases, to be replaced by uncontrollable retching; this is very characteristic. Inability to swallow now occurs, any fluid taken by mouth being immediately rejected unaltered. Distension, particularly of the epigastric and hypochondriac areas, soon commences and increases progressively. It may be enormous.

Attempts at passing a stomach tube in an endeavour to relieve the distension are invariably unsuccessful once the condition is well defined. (Siegel was able to pass a tube early, but the distension reappeared rapidly after withdrawal of the tube.) A sense of oppression and even pain in the left side of the thorax has been noted by several writers, while displacement of the apex beat to the left is also mentioned as a suggestive sign. The upper abdomen appears greatly distended in the epigastric and umbilical zones, while the iliac fossæ are comparatively flat. It is uncommon to delimit a definite swelling. The area is highly tympanitic on percussion, but a change in the percussion note on alteration of position has been described in a number of cases. Tenderness is not marked, though pain may be extreme.

Partial Volvulus.—In practically every case there is a history of old-standing digestive disturbance. There may have been several crises of severe epigastric pain, lasting a few hours and disappearing spontaneously, before the onset of the present condition. The onset is sudden, with severe pain and repeated vomiting of watery fluid mingled with bile. The stomach tube passes in many cases and may withdraw a quantity of fluid without relief.

These cases are not necessarily progressive to a fatal ending, as cardiac and even pyloric occlusion may not be absolute—the symptoms are then rather those of stenosis (vomiting at intervals; loss of weight, etc.). The history of a crisis of pain and vomiting rapidly followed by symptoms suggestive of stenosis might suggest the diagnosis. In subacute cases of this type X rays have been brilliantly successful in a few cases in making a pre-operative diagnosis.

DIFFERENTIAL DIAGNOSIS.

In the typical acute total volvulus of the stomach, a classical triad of symptoms is seen: (1) Vomiting once or twice, ceasing and being replaced by uncontrollable retching without result; (2) Rapid increasing distension in the epigastric and hypochondriac areas, the iliac fossæ remaining comparatively flat; (3) Inability to secure the passage of a stomach tube.

Even when these classical signs and symptoms are absent, the condition should be differentiated from most of the common abdominal emergencies, for example :—

Perforated Viscus.—Hyperacute onset ; tense rigidity ; tenderness ; distension not marked early.

Intestinal Obstruction.—Biliary and later faecal vomiting ; visible peristalsis in many cases.

Acute Pancreatitis.—Terrible epigastric pain, also felt in back ; persistent vomiting ; abdomen tender ; cyanosis ; less marked distension.

Mesenteric Embolism.—Vomiting ; distension ; cardiac lesion.

With regard to the cases of partial volvulus, the diagnosis becomes more difficult, as symptoms are not so definite. With patients that are not desperately ill, X rays are often of great service.

A sudden change in the symptomatology, which may have been present for years suggesting peptic ulcer or pyloric stenosis, especially if there have previously been similar crises, urgently demands exploration. By the symptoms it produces, gastric volvulus permits of no undue delay in instituting treatment whether diagnosed pre-operatively or not.

TREATMENT.

This is essentially surgical. Laparotomy must be undertaken. When the abdomen is opened the condition present must be appreciated accurately. Owing to the distortion of the normal anatomical boundaries this may be difficult ; the gastro-epiploic veins are then useful landmarks.

The next step is the reduction of the volvulus. In the majority of cases it has been found impossible to do this without first evacuating the gas and fluid content from the distended stomach. Aspiration with trocar and cannula is therefore usually practised through the posterior wall of the stomach (which is presenting), the mesentery being torn through if necessary. When the aspiration is concluded, it is important to close the opening in the stomach wall at once, otherwise it may recede so deeply in the process of untwisting as to render suturing very difficult or impossible.

The volvulus is then reduced. The detorsion is not complete until the coronary vessels are in their normal anatomical relations. In many cases this concludes the procedure in the acute stages ; the volvulus has apparently little tendency to recur immediately. Should the general condition of the patient be satisfactory, gastropexy of the Perthes type is advisable.

In partial cases, especially those associated with pyloric stenosis or hour-glass stomach, gastro-enterostomy has been performed with success.

CASES FROM THE LITERATURE.

The following is a short résumé of the published cases :—

PRE-OPERATIVE PERIOD.

Berti (1866).—Post-mortem case. Female, age 60. Sudden acute onset. "When the entire mass of organs, including the spleen and the pancreas, had been made to make two complete turns horizontally from left to right, each viscus was

restored to normal position and the channels once more became patent." (Free translation.) Berti also mentions that the œsophagus and duodenum were interlaced.

Mazotti (1874).—Post-mortem case. Female, age 50. Much preceding gastric trouble. Vertical volvulus of pyloric sac of an hour-glass stomach with some adhesions to the abdominal wall—almost a complete turn.

Dini (1879).—Female, age 42. Much preceding gastric trouble. Lived four days after acute onset. Volvulus of mesenterio-axial type—pylorus approximated to cardia. Ulcer found on posterior wall of untwisted stomach. Advanced ptosis. Spleen mobile and found in pelvis.

Langerhans (1888).—Female, age 47. Much gastric trouble. Lived five days after acute onset. Volvulus of a pyloric sac vertically round a horizontal axis. Perigastritis; sac covered by mesocolon, a loop of colon being carried up with the sac. Complete closure of pylorus by the twist.

Saakes (1903).—Female, age 67. Previous gastric symptoms. Post-mortem: Cardiac and pyloric ulcers. "The cardiac portion is well placed, but the right convex half is twisted forwards round a vertical axis so that the pylorus appears pressed closely against the cardia, which is to the right and behind it." In other words, typical mesenterio-axial volvulus.

OPERATIVE PERIOD.

Berg (1895).—Male, age 41. Pain after meals for some time. Acute onset: sound obstructed. Organo-axialis type: infraecolic. Reduction after tapping and closure—recovery.

Berg (1896).—Female, age 45. Two previous crises (lasting twenty-four hours). Onset four days before seen. Vomiting ceased after two days; pain persisted; inability to swallow; abdominal swelling. Operation after sixteen days. Partial volvulus associated with internal hernia through rent in gastro-hepatic omentum: reduction—recovery.

Wiesinger (1901).—Male, age 41. Always well. Large meal—sudden onset; vomiting, then ceased; distension and pain continued; obstinate constipation. Operation four days after onset. Blood-stained fluid and some fat necrosis in peritoneal cavity: appeared to have a tumour behind stomach; aspirated four litres of fluid; tumour disappeared. Stomach turned 180° and fixed by recent adhesions—mesenterio-axialis type. Colon above lying beneath liver (how much not stated). Both cardia and pylorus were closed. Closure of aspiration wound; reduction—recovery.

Dujon (1903).—Male, age 5. Laparotomy for acute gastric symptoms. Died in twenty-four hours. Dilated stomach reached to pelvis. It was twisted 180° around the lesser omentum so that the posterior surface came to the front. So great was the distension that both the gastro-phrenic and gastrocolic omenta were torn. Probably mesenterio-axial type.

Pendl (1904).—Male, age 63. Good health. Large meal—acute attack; rapid distension. At operation swelling was found covered with omentum. Right colon distended with gas, extended from right side and lower part of tumour upwards and backwards and disappeared to the left of the liver behind the tumour. To the left of the middle line was found the left half of the colon quite empty. Twist of 270° round a frontal axis. Emptying necessary with trocar. Marked ptosis when reduced—cure.

Borchardt (1904).—Male, age 44. Blow on abdomen twenty-four hours previously. Rapid progress of symptoms; one single vomit; obstruction; distension of epigastrium and left hypochondrium. Blood free in peritoneal cavity: splenectomy for hæmorrhage. Found twist of 180° round a horizontal axis "so that the upper and posterior quadrant lay in the pyloric region". The cardia was covered

by the pylorus: both were closed; the colon lay above the stomach. Patient died. Aortic aneurysm as a complication.

Neumann (1906).—Female, age 34. Ileus thirty-six hours. Distension; sound passed freely without result. Found a large fluid swelling, anterior surface divided towards the inferior pole by a ridge into two parts: large upper one covered by gastro-hepatic omentum, another smaller one not covered by omentum. First part represented the base of the stomach, which was twisted 180° on horizontal axis. The second was anterior stomach wall below the lesser curvature. The greater curvature was against the posterior abdominal wall. The colon lay above the volvulus. Reduction—anterior gastro-enterostomy—recovery.

Sinjuschin (1906).—Male, age 55. Good health. Sudden onset—vomited once, then retched; rapid distension. At operation a swelling was found covered by omentum; stomach twisted 180° , pylorus being lodged in front of the cardia above and to the left. Colon lay above stomach. Ptosis. Reduction—died later. Post-mortem: Ulcer near cardia and good deal of hæmorrhagic fluid in stomach.

Niosi (1907).—Female, age 38. Twenty years' history of gastric disturbance. Severe crisis before admission. Abdominal swelling noted. Sound removed considerable fasting content. Operation revealed volvulus of pyloric sac through 360° round transverse axis from pylorus to the hour-glass constriction. Congenital gap in gastro-hepatic omentum so that volvulus projected to some extent into the lesser sac. Reduction—gastro-enterostomy—cure.

Délangre (1907).—Male, age 54. Found stomach divided by furrow into two pouches—upper larger one was covered by thin layer of gastro-hepatic omentum, the smaller lying below uncovered by mesentery. The median furrow was the lesser curve, the big pouch the posterior wall, and the small pouch the anterior stomach wall partially twisted. Organo-axialis anterior.

Tuffier (1907).—Male. Previous gastric trouble. At operation many old adhesions. Chronic twist of pyloric end of stomach: easily reduced; very marked gastric hypertrophy. Gastro-enterostomy. Died.

Hermes (1908).—Female, age 66. Thirty years' gastric trouble. Crisis ten years before—cured by abdominal massage. Second crisis: admitted to hospital. Two swellings seen in stomach; much distension, but iliac fossæ were flat. Gastric lavage removed one litre of brownish fluid. Tumour decreased slightly in size. Operation revealed a large tumour descending into the pelvis. Found to be volvulus in the pyloric pouch of an hour-glass stomach around a horizontal axis, which had slipped through a breach in the transverse mesocolon. Gastric fistula was made to pyloric pouch after reduction—recovery.

Tyrmos (1909).—Male, age 53. Old gastric trouble. Sudden epigastric pain; rapid distension: vomiting. Volvulus of pyloric end of stomach (mesenterio-axial type) covered by thin omentum. Colon lying above volvulus, loaded with seybala and adherent to stomach and liver. Fair amount of perigastritis. Ulcer on lesser curvature. Gastro-enterostomy—cure.

Payer (1909).—Male, age 59. Old history of "cramps without vomiting". History of effort one and a half hours after meal. Epigastric pain and cardiac oppression: sinistrocardia (confirmed by X ray): some distension; swallowing difficult and finally impossible; gastric sound negative. Diagnosis made pre-operatively. Laparotomy seven hours after onset. Gastric volvulus 180° in forward direction (mesenterio-axial). Pylorus and cardia closed. Reduced with difficulty without trocar. Marked gastropptosis. Died twenty-four hours later.

Payer quotes 500 cases of diaphragmatic hernia with 12 gastric volvi.

Jiano (1910).—Male, age 70. Acute illness two days; no vomiting; bowels not open: marked epigastric distension. Laparotomy revealed volvulus of mesenterio-axial type. Left half of volvulus covered by colon, right half by gastrohepatic ligament. Reduced without aspiration.

Hedlung (1911).—Female, age 36. Previous gastric trouble. Partial volvulus in pyloric sac of hour-glass stomach found at operation; complicated by gastric ulcer. Volvulus estimated at 360°. Reduction was easily accomplished. Patient died.

Krymholtz (1911).—Female, age 3½. Acute onset: frequent and persistent vomiting; epigastric pain; distension. Sound passed with difficulty, nothing withdrawn. Laparotomy at thirty-six hours. Found organo-axialis type rotated through 180°; transverse colon below and normal in position. Replacement with ease—cure.

Kerr (1912).—Male, age 40. Previous gastric symptoms. Two previous crises each following a heavy meal, relieved by self-induced vomiting and abdominal massage. Sudden onset; severe epigastric pain; no vomiting even with apomorphine; negative result with a stomach tube; great epigastric distension; sinistocardia. Laparotomy by two-inch incision revealed stomach not covered with omentum. This was aspirated and much gas and 54 oz. of fluid were withdrawn. Closure of abdominal wound (case being taken for one of acute gastric dilatation). Collapse fourteen hours after operation. Post-mortem examination showed organo-axialis type with the great omentum torn away from the greater curvature and colon lying below the twisted stomach. Nothing grossly pathological was found in the stomach.

Bourcart (1913).—Female, age 52. Previous gastric trouble for thirty-six years. Marked ptosis; crises of abdominal pain. X ray—bilocular stomach reported. Was actually in volvulus when X-rayed, but not recognized as such. Laparotomy revealed no definite adhesions. Chronic twist in pyloric sac of hour-glass stomach with a good deal of compensatory hypertrophy. Gastroplasty.

Kocher (1914).—Female, age 53. Previous gastric symptoms. Developed acute attack while in hospital under observation. Sudden onset: severe pain, epigastric and in the back; retching; rolling about in bed. Abdomen distended in upper half; swelling in epigastrium, resonant above, and dull below. Stomach tube withdrew 1500 c.c. of sour fluid. No gas removed by tube. Early collapse. Laparotomy at two and a half hours. Found mesenterio-axialis type in an hour-glass stomach (unique case). Twist estimated at 270°. Some chylous fluid in pelvis (? lymphatic obstruction). Reduction easy. Marked ptosis. Hour-glass stomach. Closure. Made slow recovery hindered by marked ptosis and delay in emptying. Gastro-enterostomy as second-stage operation. Recovery.

Kocher quotes von Harberer (1912), and mentions two early cases of Colliclion.

Siegel (1920).—Female, age 2. Acute abdominal symptoms. Stomach tube withdrew quantity of watery fluid. Rapid re-accumulation of fluid together with distension allowed of pre-operative diagnosis. Operation—recovery.

Rosselet (1920).—Female, elderly. The first case of complete volvulus observed by X rays. Apparently symptomless. The patient was under observation for chronic sigmoid obstruction. At first examination a most peculiar radiogram was obtained (reproduced in Rosselet's paper); there was not complete obstruction of the cardia and pylorus. At a later date further radiograms were taken, but showed a normal stomach outline. There was much ptosis. Exaggerated 'aërocolie' was noted at splenic flexure and was held to be the exciting cause. Laparotomy not performed.

Rosselet and Gilbert (1922).—These observers were investigating the case of a male, age 45, who had suffered for thirteen years with gastric symptoms. Several crises of severe abdominal pain with watery vomit. Always better when recumbent. During observation X-ray screening was being performed with the patient in the Trendelenburg posture. Partial volvulus of the pylorus from behind forwards in front of the fundus was noted; no apparent symptoms. Laparotomy later showed stomach in normal position. There was a chronic ulcer on the lesser curvature and some perigastritis. Gastro-enterostomy.

Thorek (1923).—Female, age 77. Acute onset; severe epigastric pains after heavy meal; vomiting at first, soon superseded by nausea and retching without result; marked distension of epigastric and umbilical areas; heart normal in position; obstinate constipation; no visible peristalsis. Laparotomy under spinal anaesthesia. Large mass presented. Tapped after raising from pelvis. Stomach found to be twisted 270° on a horizontal axis, the pylorus being dragged to the left side and the cardiac end completely twisted. The transverse colon was lying vertically and above the lesser curvature. Spleen displaced forwards. Marked ptosis found when the stomach was untwisted. Suspended by gastropexy. Gastric lavage seventy-two hours—complete recovery.

Halles (1923).—Male, age 38. Irregular crises of lower abdominal pain for years—lasting half an hour—relieved by vomiting or by standing on the head. Sudden onset three days before admitted. Severe abdominal pain; vomiting giving place to retching; bowels obstinately constipated; marked distension in epigastric area; iliac fossae flat; uncontrollable retching. Laparotomy showed stomach covered by thin veil of mesocolon. Stomach untwisted in clockwise direction after aspiration puncture. Colon lay above stomach beneath liver. Some traction on pancreas and few patches of fat necrosis. Stomach evacuated by tube for few days after operation—recovery. Later X ray showed that the fundus of the stomach reached to true pelvis.

Nockolds (1924).—Female, age 42. Old gastric trouble. Five days' vomiting—watery fluid. Pain; tenderness; absolute constipation; patient moribund when seen. Died twelve hours later. Post-mortem: Hour-glass stomach with rotation of pyloric pouch about a vertical axis from the left to right through 360° . Scar of an old healed ulcer on lesser curve.

Gill (1925).—Female, age 58. Old gastric symptoms. Three days' history of watery vomiting; no bile; distension of abdomen; visible peristalsis; not tender. Laparotomy showed volvulus of pyloric pouch of an hour-glass stomach through 360° round a vertical axis from left to right. The pouch passed through slit in gastro-hepatic omentum. Reduction—recovery. Some months later partial gastrectomy was performed with success—condition was an hour-glass stomach with two small chronic ulcers on lesser curvature.

Choisy and Babantz (1927).—Female, age 41. Gastric symptoms fifteen days—colic, distension, intestinal stasis, persistent vomiting. Partial volvulus of stomach diagnosed by X-ray examination. Three days later confirmatory X ray showed stomach in normal position. There was exaggerated mobility and the stomach was empty in two hours. At a third examination the volvulus was present and actually untwisted under the influence of the weight of barium. Laparotomy showed normal stomach with no adhesions. The writers insist that volvulus of less than 180° can exist without striking symptoms. Where there is no organic lesion of the stomach, volvulus may be the result of intestinal pneumatosis + gastric hyperkinesia. Colon may have important rôle at times. Vague symptoms may be elucidated by radiogram.

Jean (1927).—Gastric syndrome; intermittent epigastric tumour at times. X-ray examination showed stomach in partial volvulus. Pre-operative diagnosis of volvulus in an hour-glass stomach was made and confirmed at operation. Colon lay above pyloric sac, whose posterior surface was covered by mesocolon. Colopexy and gastropexy, with complete relief.

BIBLIOGRAPHY.

- BIERTI, *Gaz. med. ital. Prov. Veneti*, 1866, ix, 139.
MAZOTTI, *Riv. clin. di Bologna*, 1899, iv, 280.
DINI, *L'Imparziale*, 1879.
LANGENHANS, *Virchow's Arch.*, iii, 387.

- SAAKES, *Ibid.*, cxxxiv.
 BERG, *Nord med. Arkiv*, xiii.
 WIESINGER, *Deut. med. Woch.*, 1901.
 DUJON, *Gaz. méd. de Paris*, 1903.
 PENDL, *Wien. klin. Woch.*, 1904.
 BORCHARDT, *Arch. f. klin. Chir.*, 1904.
 NEUMANN, *Deut. Zeits. f. Chir.*, 1906, lxxxv.
 SINJUSCHIN, *Khirurgia*, 1906, No. 120.
 NIOSI, *Presse méd.*, 1908, April. Analysis from *Riv. med.*, 1907, 1287.
 DELANGRE, 20^{me} Congrès français de Chir., 1907, 286.
 HERMES, *Deut. Zeits. f. Chir.*, 1908, xciii.
 TYRMOS, *Jour. de Chir.*, 1909, May. Analysis from *Khirurgia*, 1909.
 PAYER, *Ibid.*, Dec. Analysis from *Grenzgeb. d. Med.*, 1909, xx.

The cases of all the above authors are mentioned by Tuffier and Jeanne.

- TUFFIER and JEANNE, *Rev. de Gynécol. et Chir. abd.*, 1912, No. 18, 27.
 JIANO, *Brit. Med. Jour.* (epit.), 1910, Jan. 1.
 HEDLUNG, *Jour. de Chir.*, 1911, 667. Analysis from *Nord. med. Arkiv*, xlv.
 KRYNHOLTZ, *Khirurgia*, 1911.
 KERR, *Ann. of Surg.*, 1912, Nov., 697.
 BOURCART, *Rev. de Chir.*, 1913, Nov., 800.
 KOCHER, *Deut. Zeits. f. Chir.*, 1914, cxxvii, 591.
 SIEGEL, *Zentralb. f. Chir.*, 1921, May, 618.
 ROSSELET, *Jour. de Radiol. et d'Electrol.*, 1921, iv, 341.
 ROSSELET and GILBERT, *Ibid.*, 1922, vi, 76.
 THOREK, *Jour. Amer. Med. Assoc.*, 1923, Aug., 636.
 HAILES, *Med. Jour. Australia*, 1923, Nov., 544.
 NOKKOLDS, *Brit. Jour. Surg.*, 1924, xi, 774.
 GILL, *Irish Jour. Med. Sci.*, 1925, 418.
 CHOISY and BABANTZ, *Acta Radiol.*, 1927, 410.
 JEAN, *Bull. et Mém. Soc. Nat. de Chir.*, 1927, liii, 110.

ON SOME ASPECTS OF THE PATHOLOGY OF HYPERTROPHIC CHARCOT'S JOINTS.

By E. J. S. KING,

STEWART LECTURER IN PATHOLOGY, UNIVERSITY OF MELBOURNE.

ALMOST from the inception of our knowledge of Charcot's disease of joints, the pathology has been enwrapt in the mysterious obfuscation of such terms as 'trophic disturbance' and 'neuro-arthropathy'. At first the frequent occurrence of the condition in cases of tabes dorsalis also led to the erroneous conclusion that the joint changes must be syphilitic in origin. Two different types of observations have helped to clarify the situation:—

1. There was the discovery that many forms of nervous lesions—in addition to tabes and general paralysis of the insane⁶—are associated with the characteristic joint changes of Charcot's disease. In addition to syringomyelia, paraplegia, and myelitis, peripheral nerve lesions have been shown to present the same complication.²⁸ This led to the exclusion of syphilis as an immediately causative influence.³⁴

2. The experimental work done by Eloesser¹¹ showed that the production of anæsthesia in a limb, by section of the posterior nerve-roots in animals, did not of itself produce joint changes, but that trauma to the anæsthetic joints produced typical Charcot's disease. The importance of trauma in the causation of joint changes has also been emphasized on clinical grounds.³¹

The conclusion that the phenomena are not explained by the nugatory terms 'trophic' and 'arthropathy' necessitates an investigation of the pathology in the light of the fundamental processes of inflammation and neoplasia. It is proposed to show that some at least of the changes of Charcot's joints can be dealt with in this manner, and since this investigation developed in the course of a study of other diseases of the joints, particularly osteo-arthritis, it will be convenient to consider these first.

For many years joint diseases were considered and classified purely from the clinical aspect, without reference to their pathology.³² It was thought that the pathologist did not assist clinicians to understand these conditions.²⁵ and possibly this was so. A thorough study of microscopic joint changes was made in 1909 by Nichols and Richardson,²³ and they pointed out the importance of injury in many of these cases.



FIG. 57.—Charcot's disease of the ankle, showing the extraordinary swelling of the joint.

A further advance was made in the description by Axhausen² of small pieces of dead bone in the sections of the articular ends of bones in cases of

osteo-arthritic disease. The next step in the chain of evidence was the experimental implantation of small pieces of bone in the subcutaneous tissues—the ear of the rabbit, for example.²² It was shown that this resulted in a curious reaction of the connective tissue; metaplasia occurred, with bone and cartilage formation. This observation has been repeated and confirmed.^{17,29}

These discoveries were applied experimentally to joints by Leriche,¹⁷ who implanted pieces of bone into the joints of animals. He used fresh bone and, as a control, bone that had been killed by alcohol, and both intra- and extra-synovial implants were made. The dead bone was found to be absorbed without producing any irritative effect, but the fresh bone was found, on examination after varying periods, to have died and at the same time to have caused a remarkable proliferation of the surrounding tissues. Thickening of the synovial membrane, with the formation of polypoid processes, occurred, while osteocartilaginous change took place in the neighbouring tissues. Though phenomena such as these are very suggestive, still further work is required to establish the suggested correlation. It was the discovery of changes similar to those found in the cases of



FIG. 58.—Sagittal section of the ankle-joint, showing the destruction of the joint and the sequestrum (A) in the astragalus.

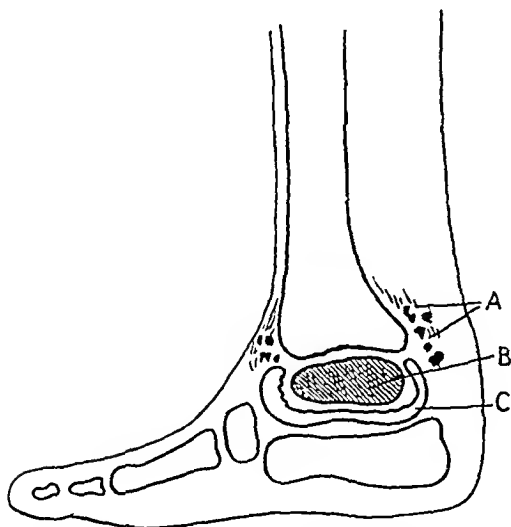


FIG. 59.—Diagram of ankle-joint shown in Fig. 58. A, Pieces of bony and cartilaginous tissue; B, Sequestrum; C, Shell of astragalus.

osteo-arthritis, as well as to those produced in experimental work, that led to a study of some aspects of Charcot's disease and to deductions concerning the pathological processes involved.

Of six specimens, the first encountered showed the most striking alterations (*Fig. 57*). The appearance of the leg when sectioned after amputation is shown in *Figs. 58* and *59*. There was loss of cartilage and roughening of the lower end of the tibia seen characteristically in advanced joint diseases of this kind. The articular surface was irregular, uneven, and somewhat bluish in colour, but differed from normal cartilage in having a more 'opaque' appearance. The astragalus was greatly enlarged, and consisted of a thin-walled cup of bone containing a large sequester (*Figs. 58, 59*). On the surface in contact with the sequester this shell or cup was eburnated and partially covered by cartilage. This cartilage was similar to that found on the lower aspect



FIG. 60.—Photomicrograph of portion of the tissues adjacent to the joint. A small piece of degenerated bone (a) is present, and, in close proximity, active fibrous tissue with cartilage (b) and bone (c) formation is seen. ($\times 50$.)



FIG. 61.—Photomicrograph of the joint surface of the tibia. No original cartilage remains, the tissue lining the bone (a) being fibro-cartilage and fibrous tissue with vessels running at right angles to the joint surface. ($\times 40$.)

of the tibia, and immediately attracted attention, since it was lining, not the original joint surface, but a newly formed cavity made by the separation of the sequester from the astragalus. The mid-tarsal and sub-astragaloid joints appeared to be normal.

Microscopic section of the tibia and astragalus showed varying pictures in different parts:—

1. In some areas small fragments of dead bone were present (*Fig. 60*). Some of these were in continuity with the bony tissue, while others were situated in the surrounding connective tissue. In some portions of bone the tissue of the Haversian canals had become necrotic. The vessels and the fibrous tissue had become granular and degenerated. The bony tissue immediately surrounding these spaces stained differently from the bony trabeculae further removed, but whether this was due to anæmia and death of

bone was not definitely determined. The cause of the death of some areas of bone, whether from the cutting off of the blood-supply or not, was uncertain, but the occurrence of broken-down granular bony tissue was undoubted.

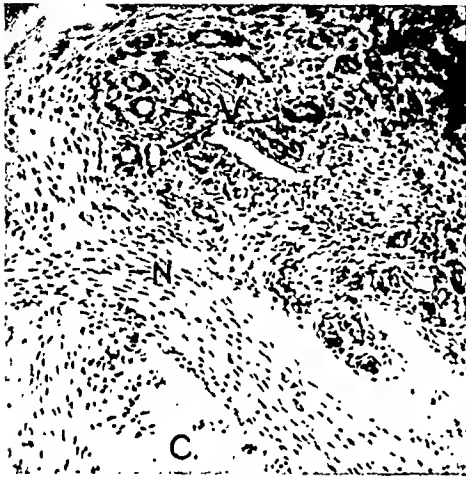


FIG. 62.—This shows a typical appearance in many of the areas—active connective tissue (N), well-formed blood-vessels (V), and recently laid down cartilage (C). ($\times 110$.)



FIG. 63.—Showing evidence of bone absorption. The Haversian canals are widened and the lamellae have been irregularly eroded. Vascular dilatation is in evidence. ($\times 95$.)

2. There was marked activity of the cells of the tissues in many parts of the sections (Fig. 61). This activity consisted of a great deal of fibroblastic proliferation and a large development of bony and cartilaginous tissue. In some places this was particularly marked in relation to pieces of necrotic bone.



FIG. 64.—In some areas there were numbers of phagocytic cells containing blood-pigment. ($\times 110$.)

One striking feature was the great variety of the appearances presented by the bone and cartilage in these areas. The sections were stained by hæmatoxylin and eosin, iron hæmatoxylin and van Gieson, and by Schmorl's stains. A large number of bony trabeculae were abnormally calcified; in some parts only a small amount of normal bone was present. Those portions which were normal in form showed great variation in staining power, thus rendering an accurate interpretation of the actual state of any particular

portion extremely difficult. Some variation was due to new formation—the

age of the ossein being one cause of the variety of staining capacity—while other portions were undergoing absorption. Some of the cartilage had a large number of cells in a small area, while other parts had extremely few cells in a large mass of tissue (*Fig. 62*). Some portions showed direct transition from this tissue into bone. While the greater part of the cartilage was hyaline, that portion which was near the cavity of the joint was fibrocartilage.

In the proliferating areas the vessels were very well developed, showing completely formed walls (*Fig. 62*). The change was of such a degree as to suggest a neoplastic rather than an inflammatory vascular proliferation.

3. Any microscopic evidence of syphilis was strikingly absent. The fibrous tissue in the Haversian canals was of the osteoid type seen in some bone diseases, and was quite unlike the fibrosis of syphilitic inflammation. The vessels of the Haversian canals also were thin-walled, showing none of the endothelial and fibroblastic proliferation so characteristic of luetic inflammation. As noted above, where the proliferation was most marked the vascular increase was of fully formed vessels, but these



FIG. 63.—Typical Charcot's disease of the knee-joint, showing osteophytic growths and development of bone in a tendon.



FIG. 66.—Typical radiogram of Charcot's disease. The tremendous new formation of bone in the tissues around the joint is well seen. There is also atrophy of the tibia.

did not show excessive thickening. (*Fig. 63.*)

Throughout the sections the presence of a large number of cells containing blood-pigment gave an indication that hæmorrhage—and possibly trauma—had occurred, with phagocytosis of the blood-pigment (*Fig. 64*). The irregular distribution suggested that this was traumatic in origin rather than due to a general vascular disturbance.

4. That the new growth of bone was associated with architectural remodelling was shown by

decalcification of parts, with or without the activity of osteoclasts. In some portions the bone nearest the fibrous tissue of the Haversian canals showed a difference of staining power and a gradual change to osteoid tissue. Osteoclasts were present in places, but in others were completely absent, halisteresis apparently occurring in these areas. In the large masses of new bone Volkmann canals were abnormally numerous throughout the tissue, these canals having no relation to the structure of the lamellæ of the trabeculæ.

The five other cases examined have shown the same type of change in the tissues in the region of the joints. These comprised two knee-joints, an ankle, an elbow, and a hip-joint. The degree of change varies considerably in different cases. In one case—a knee—the pathological tissue was much less active, and resembled adult tissue to a much greater extent, than in the other cases. At the same time the macroscopic changes were much less striking. In the case of the other knee, in which there were growths of bone into the surrounding muscles and tendons (*Figs. 65 and 66*), the number of phagocytic cells containing blood-pigment was particularly large.

DISCUSSION.

The rapidity of the progress of Charcot's joints, the grotesque nature of the deformities, and the associated nervous lesions apparently have obscured the judgement of investigators to such a degree that special pathological processes have been invoked to explain the phenomena. This study was undertaken to show that a critical examination would enable some at least of the processes involved to be reduced to terms of those fundamentals by which we explain other conditions. It is necessary first to consider the physiological and simpler pathological relationships of the cells with which we are to deal.

A study of the growth of bone in the embryo shows that the formation of cartilage and bone are manifestations of the multipotentialities of the mesenchymal cell.³⁵ Some of these cells produce cartilage,^{15, 18} while at a later stage, under different influences, similar cells produce osseomucin or ossein and so provide the more resistant tissue—bone. Still others produce the reticular cells of the marrow and the hæmoblasts. In normal circumstances, therefore, as the organism ages the cells become more and more differentiated and specialized.

In the embryo a reversion to the primitive type can readily occur, but as the body develops, the potentialities of the cell for anaplastic proliferation gradually diminish.⁹ However, these potentialities are merely latent, as is shown, for example, by the occasional development of bone-marrow tissue in the adult kidney.¹⁹

The conception of the important place of the primitive connective-tissue cell as the progenitor of all mesoblastic cells has not been recognized adequately, so that varying accounts of the development of tissues have been given. The growth of osteoblasts from fibroblasts,³⁶ and of blood-cells from endothelium, are merely other and less complete expressions of the same idea.

When we turn to pathological processes, we frequently find exciting factors causing the reversion of adult cells (de-differentiation) to the original embryonic

—and therefore multipotential—type. These cells ultimately undergo differentiation once more, with the formation of an allied though different form of cell. The frequency with which this 'metaplasia' of the connective tissues throughout the body occurs is reflected in the literature. Formation of bone in diseased vessel walls has been described, and calcified material has been given as the exciting influence producing these changes.⁷ Bone tissue containing red marrow has occurred in the same situation.⁸ Experiments have demonstrated the production of bone in kidneys,^{27, 33} and bone has even been found in a growth in the skin.²⁴ The formation in many other parts has been reported.³⁰

Cell proliferation and metaplasia do not occur only in the case of the simpler connective-tissue cells—the fibroblasts. Originally it was thought that the osteoblast could merely nourish a certain amount of surrounding bone, and that further growth—the result of the stimuli of trauma or infection—came from the cells of the periosteum. It has been shown, however, that the periosteum is merely a limiting membrane,^{16, 20} and that in certain circumstances the bone-cells can resume their more primitive character of multiplication. The possibilities of cellular activity in the case of the bone-marrow have been shown experimentally.²¹ Thus, all the tissues taking part in the formation of bone may, in suitable conditions, become more 'embryonal' and multiply. It is necessary, therefore, in the study of any pathological process involving cells of many potentialities, to take into consideration the possibilities, both of direction and degree of growth, if we would understand the gross features which are presented to us.

The changes of the simple fracture exemplify the whole process. Here the stimulus is, apparently, the presence of disintegrated and autolysed cell material. This causes an intense proliferation of the connective-tissue cells and bone-cells, with the subsequent formation of osteoid tissue and, ultimately, bone—cartilage being an intermediate stage. Occasionally the formation of cartilage may be so gross as to be apparent macroscopically. It has been remarked that, microscopically, such callus may resemble very closely an osteogenic sarcoma. This very observation gives the key to the processes which are being discussed. Anaplasia of the cells has occurred, and the majority of the cells present, at some period or other, have been of the embryonic type. It is not proposed to discuss the difficult question of the relationship of repair processes to neoplasia, but it can readily be seen that the relationship between the two in the active stages of normal repair is close.

If we now apply these ideas to a study of the changes observed in Charcot's joints, a similarity is immediately apparent (*Figs. 67-69*). There is some factor which is causing proliferation of the cells with a reversion to the primitive form. This stimulus causes a release from the inhibitions imposed by their normal relationship to all the surrounding cells. Later there occurs a development of a different order, a second differentiation.

It is suggested that the stimulus is the presence of small pieces of dead bone in the tissues. This is not proven, but the discovery of these pieces of bone in close relationship to the areas of cellular activity, together with the results of the experimental work previously referred to, is strong circumstantial evidence.

The cause of the necrosis of bone is, presumably, multiple injuries, which are possible since the joints are anæsthetic. Pain has been experienced in some cases,¹² but this does not negative the thesis, since the pain is due to the stretching of the skin, and sensory impressions from the skin and deeper structures are conveyed in different tracts.

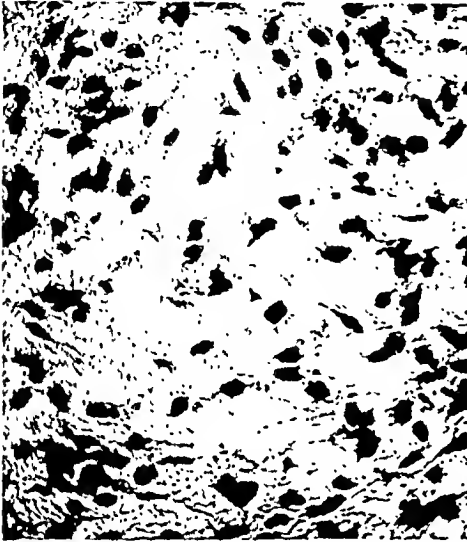


FIG. 67.—An area of proliferating connective tissue. The resemblance to a connective-tissue neoplasm is apparent. ($\times 345$.)



FIG. 68.—Photomicrograph showing some of the proliferating tissue. The formation of connective tissue and bone is well seen. ($\times 95$.)

The metaplasia of the various connective tissues is not confined to a formation of bone- and cartilage-cells, since some of the bony outgrowths at a distance from the original bone contained marrow (Fig. 70), thus indicating further the multipotentialities of the cells. Further activity of the marrow was shown in the presence of 'fœtal' fat cells amongst the hæmopoietic cells. It is still undecided whether the embryonic fat cell—steatoblast—arises directly from the mesenchymal cell or as a modification of the fibroblast; but in either case, as stated previously, these are merely variants of the



FIG. 69.—Showing the metaplasia to bone and cartilage. ($\times 100$.)

one idea. Cellular activity and anaplasia are in this case beyond doubt (Fig. 71).

The activity also extends to the vessels (Fig. 72). There is considerable vascular proliferation, and in some portions the vessels are surprisingly well formed, suggesting a neoplastic rather than inflammatory element. Incidentally it has been pointed out that, in osteo-arthritis, some of the proliferative changes are midway between inflammation and neoplasia.¹³ The cells proliferate rapidly and in an apparently uncontrolled fashion. Presumably these phenomena are due to the repetition of the exciting stimuli—injury and necrosis of small pieces of bone. Frequently bony growth occurs, not only around the joint, but into and along the muscles and tendons in relation to the joint.³

Bone Absorption.—A striking feature is the absorption of bone, which apparently maintains the architectural structure of the new tissue. Some absorption of the



FIG. 70.—A small piece of bone at a distance from the original bony tissue. The formation of cartilage, bone, and bone-marrow is apparent. ($\times 45$.)



FIG. 71.—An area of the new formed tissue showing activity of marrow, which is forming largely connective-tissue and fat cells. ($\times 45$.)



FIG. 72.—The cellular tissue adjacent to the bone, showing the vascular activity. ($\times 100$.)

original bone can also be observed. Bone removal occurs in three ways:¹⁰ (1) *Halisteresis*; (2) *Lacunar erosion*; and (3) *Vascularization* by means of perforating Volkmann canals.

1. *Halisteresis*.—Kilian¹ first used the term to describe the softening of bones in osteomalacia. The basis of the process is the absorption of calcium by means of the body fluids. The reasons for considering that this occurred in the cases examined were that there was an area of 'osteoid' in the Haversian canals, although no osteoclasts were present. The osteoblasts in the surrounding bone were lying also in clear zones that were larger than the normal lacunæ.

It has been considered that both the building up and the disintegration or absorption of bone is under the control of the osteoblasts.⁵ The processes of the osteoblasts seemed to be shortened in the areas of halisteresis, but the relationship between the microscopic alterations in the cells and the absorption of bone was indeterminate.



FIG. 73.—Erosion of bone by means of osteoclasts. A lacuna (L) is present, but the impression given is that the cell activity is secondary to some other influence. (See text.) ($\times 150$.)



FIG. 74.—Area of new formation with vascularization by Volkmann's canals. In some of these degeneration has occurred in the tissue, this degeneration being similar to that seen in a few of the Haversian canals. ($\times 170$.)

2. *Lacunar Erosion* (Fig. 73).—It has been held for a considerable time that osteoclasts cause destruction and resorption of bone. It is probable, however, that they act quite secondarily. Resorption is often too rapid to be due to osteoclasts. They probably act like foreign-body giant cells elsewhere. Osteoclasts were numerous in some sections examined, though absent in others.

3. *Vascularization by Means of Perforating Volkmann Canals*.—Resorption by perforating vessels occurred extensively in the areas of new bone formation. Apparently they aided the remodelling of the texture. This form was not

observed in the areas of rarefaction of the original bone. The microscopic appearances of the perforating and the Haversian canals are quite characteristic and distinct, in that the former have no relationship to the lamellæ of the bone (*Fig. 74*).

The ultimate processes by which the rarefaction of the bone is caused are at present obscure; but when more is known of the physiology of the sympathetic supply of the blood-vessels and their reaction to trauma, these processes will probably be placed on a sound and simple basis.

CONCLUSION.

As far as the hypertrophic changes of Charcot's joints are concerned, it is suggested that the complicated phenomena of the disease may be resolved into the simple principles of pathology—the stimulation of the cells of the part to proliferation, and the secondary differentiation with the formation of excess connective tissue, cartilage, and bone tissues.

SUMMARY.

1. Hypertrophic Charcot's disease is a grotesque form of osteo-arthritis, and the exaggeration of the processes concerned is due to the more frequent traumatisms which are permitted by the anæsthetic condition of the bones and joints.

2. The processes involved are anaplasia of the connective-tissue cells to a primitive type, under the action of some stimulus, with subsequent differentiation in various directions.

3. The phenomena may be of such an anaplastic order as microscopically to suggest neoplasia.

4. It is suggested that the stimulus is the products of dissolution of small pieces of necrotic bone.

BIBLIOGRAPHY.

- ¹ ANHAUSEN, G., "Arbeiten aus dem Gebiet der Knochenpathologie und Knochenchirurgie", *Arch. f. klin. Chir.*, 1911, xciv, 241.
- ² ANHAUSEN, G., "Die aseptische Knochennekrose und ihre Bedeutung für die Knochen- und Gelenkchirurgie", *Acta Chir. Scand.*, 1926, lx, 369. Quoted by Leriche.
- ³ BARTH, "Histologische Knochenuntersuchung bei tabischer Arthropathie", *Arch. f. klin. Chir.*, 1903, lxi, 174.
- ⁴ BAST, T. H., "Studies on the Structure and Multiplication of Bone Cells Facilitated by a New Technique", *Amer. Jour. Anat.*, 1921, xxix, 139.
- ⁵ BAST, T. H., "A Note on the Phagocytic Activity of Bone Cells", *Anat. Record*, 1924, xxviii, 91.
- ⁶ BILLINGTON, W., and BARNES, A. S., "Charcot's Disease of the Ankle in a Case of General Paralysis of the Insane", *Lancet*, 1905, ii, 23.
- ⁷ BUEGER, L., and OPPENHEIMER, A., "Bone Formation in Sclerotic Arteries", *Jour. of Exper. Med.*, 1908, x, 354.
- ⁸ BUNTING, C. H., "The Formation of True Bone with Cellular (Red) Marrow in the Sclerotic Aorta", *Ibid.*, 1906, viii, 365.
- ⁹ DANCHAKOFF, V., "Cell Potentialities and Differential Factors, considered in Relation to Erythropoiesis", *Amer. Jour. Anat.*, 1918, xxiv, 1.
- ¹⁰ DAWSON, J. W., and STRUTHERS, J. W., "Generalised Osteitis Fibrosa", *Edin. Med. Jour.*, 1923, xxx, 421.

- ¹¹ ELOESSER, L., "On the Nature of Nenropathic Affections of Joints", *Ann. of Surg.*, 1917, lxxvi, 201.
- ¹² ELOESSER, L., "A Sign Occurring in Cases of Tabes Complicated by Charcot's Joints", *Jour. Amer. Med. Assoc.*, 1921, lxxvii, 604.
- ¹³ FISHER, A. G. T., "Some Researches into the Physiological Principles Underlying the Treatment of Injuries and Diseases of the Articulations", *Lancet*, 1923, ii, 541.
- ¹⁴ GALLIE, W. E., and ROBERTSON, D. E., "Repair of Bones", *Brit. Jour. Surg.*, 1920, vii, 211.
- ¹⁵ GEDDES, A. C., "Origin of Osteoblasts and Osteoclasts", *Jour. Anat. and Physiol.*, 1913, xlvii, 159.
- ¹⁶ KEITH, A., and HALL, M. E., "Specimens of Long Bones Showing the Processes of Infection and Repair", *Brit. Jour. Surg.*, 1920, vii, 302.
- ¹⁷ LERICHE, R., and BIENCKMAN, E., "Recherches expérimentales sur le Mécanisme de Formation de la Chondromatose articulaire et de l'Arthrite déformante", *Presse méd.*, 1928, xxxvi, 1441.
- ¹⁸ MAIL, F., "On the Development of the Connective Tissues from the Connective-tissue Synectium", *Amer. Jour. Anat.*, 1921, xxix, 329.
- ¹⁹ MAXIMOV, A., "Experimentelle Untersuchungen zur postfötalen Histogenese des myeloiden Gewebes", *Beitr. z. pathol. Anat.*, 1907, xli, 122.
- ²⁰ MORLEY, J., "Traumatic Myositis Ossificans Resulting from Gunshot Wounds", *Brit. Jour. Surg.*, 1920, vii, 178.
- ²¹ MULLER, G. L., "Experimental Bone-marrow Reactions", *Jour. of Exper. Med.*, 1927, xlv, 390.
- ²² NAGROTTI, J., "Formation de Pièces squelettiques surnuméraires, provoquée par la Présence de Greffons morts dans l'Oreille du Lapin adulte", *Comptes rend. Soc. de Biol.*, 1919, xxxi, 113.
- ²³ NICHOLS, E. H., and RICHARDSON, F. L., "Arthritis Deformans", *Jour. Med. Research*, 1909, xxi, 149.
- ²⁴ NICHOLSON, G. W., "The Formation of Bone in a Calcified Epithelioma of the Skin, with some Remarks on Metaplasia", *Jour. Pathol. and Bacteriol.*, 1917, xxi, 287.
- ²⁵ PAINTER, C. F., "Types of Rheumatoid Disease", *Boston Med. and Surg. Jour.*, 1904, cli, 538.
- ²⁶ PEARBODY, F. W., "A Study of Hyperplasia of the Bone-marrow in Man", *Amer. Jour. Pathol.*, 1926, ii, 487.
- ²⁷ PEARCE, R. M., "Notes on the Later Stages of Repair of Kidney Tissue (Dog), with Special Reference to Proliferation of the Pelvic Peritoneum and Heteroplastic Bone Formation", *Jour. Med. Research*, 1909, xx (N.S. xv), 53.
- ²⁸ PHILIPS, H. B., and ROSENHECK, C., "Neuroarthropathies of Peripheral Nerve Origin", *Jour. Amer. Med. Assoc.*, 1926, lxxxvi, 169.
- ²⁹ POLATTINI, B., "Su Neofornazioni cartilaginee ed ossee determinate da Frammenti di Cartilagine e d'Osso fissati", *Arch. ital. di Chir.*, 1922, vi, 179. Quoted by Leriche.
- ³⁰ POSCHAUSSKY, J. F., "Ueber heteroplastisches Knochenbildung. Eine pathologisch-histologische und experimentelle Untersuchung", *Beitr. z. pathol. Anat.*, 1905, xxxviii, 596.
- ³¹ POTTS, W. J., "The Pathology of Charcot Joints", *Ann. of Surg.*, 1927, lxxxvi, 596.
- ³² RICHARDSON, F. L., "A Clinical Report of Seventy-five Cases of Arthritis Deformans (Chronic Non-tuberculous Arthritis)", *Boston Med. and Surg. Jour.*, 1905, clii, 263.
- ³³ SACERDOTTI, C., and FRATTIN, G., "Ueber die heteroplastische Knochenbildung", *Arch. f. pathol. Anat. u. Physiol.*, 1902, clxviii, 431.
- ³⁴ STANGARDT, K., "Ueber die Aetiologie der tabischen Arthropathien", *Arch. f. Psychiat.*, 1912, xlix, 936.
- ³⁵ STUMP, C. W., "The Histogenesis of Bone", *Jour. of Anat.*, 1925, lix, 136.
- ³⁶ TODD, T. W., "A Preliminary Communication on the Development and Growth of Bone and the Relations thereto of the Several Histological Elements Concerned", *Jour. and Anat. Physiol.*, 1913, xlvii, 177.

A STUDY OF TUMOURS AND INFLAMMATIONS OF THE GASSERIAN GANGLION.

By F. A. R. STAMMERS,

ASSISTANT SURGEON TO THE GENERAL HOSPITAL, BIRMINGHAM;
SURGEON TO OUT-PATIENTS, CHILDREN'S HOSPITAL, BIRMINGHAM.

TUMOURS of the Gasserian ganglion, though of sufficient rarity and interest to merit publication, can no longer be looked upon as pathological 'freaks', since in the past twenty-five years about sixty cases have been reported. Inflammatory lesions of the ganglion on the other hand are extremely rare—indeed, I can find the reports of but three examples definitely proved to be of an inflammatory nature, though several cases of mastoiditis exhibiting symptoms pointing to involvement of the ganglion have been described.

This paper is based on seven cases of neoplastic, and two of inflammatory, lesions of the Gasserian ganglion. They represent all the instances of this nature that can be traced in the records of the Mayo Clinic. Four of the tumour cases have already been reported by Dr. Sheldon,¹ Head of the Neurological Department at the Mayo Clinic, and I am indebted to him for allowing me to include them here in order to make the series complete. Fresh sections have been prepared for this work. At the time when the youth with the pituitary tumour and also *Case 7* were investigated and treated at the Clinic, I was acting assistant to Dr. Adson, so was able to follow these cases throughout their sojourn in hospital. I have also seen and examined *Case 9*, though this, of course, was post-operatively.

For the sake of clearness this paper is treated under several headings: (I) *Development of the Gasserian ganglion*; (II) *Anatomy*; (III) *The tumour cases with a discussion of the literature*; (IV) *The inflammatory cases with a discussion of the literature*; (V) *Discussion on the pathology*; (VI) *Operations*; (VII) *Differential diagnosis*; (VIII) *Conclusions*.

The development of the ganglion is briefly described because of its significance in relation to the possible sources of origin of tumours, and also in connection with Frazier's operation of subtotal resection of the sensory root for the tic douloureux. This operation will be discussed under the appropriate heading. The anatomy of the Gasserian ganglion receives such cursory consideration in most text-books, and yet has such an intimate association with symptoms and signs following on a lesion in this region, that it is described fairly extensively. Though the literature has been fully studied, it is not the purpose of this paper to make a meticulous analysis of all the cases reported up to date. Sufficient references are made to show a certain similarity in these cases, and from this similarity to develop a suggestive symptom-complex. In considering the pathology one is faced with the difficulty of nomenclature. An attempt to work on a histogenetic basis has been made, though when dealing with so-called endothelial cells difficulties arise from the very

beginning. For purposes of comparison a normal ganglion has been studied and its features emphasized. Microphotographs are presented. As diagnosis in these lesions, though highly suggestive, is never certain until the ganglion has been exposed, the operation always begins as if for treatment of tic douloureux. The various types of operation are therefore discussed. The question of differential diagnosis involves not only other intracranial lesions in the region of the Gasserian ganglion, but the whole problem of pain in the area of distribution of the Vth cranial nerve. These matters are considered, and illustrative cases are drawn from literature and personal experience.

I. DEVELOPMENT.

At the fourth week of intra-uterine life, that is, in a 4-mm. embryo, the Gasserian ganglion is represented by a continuous, compact, oval mass (Bartelmez and Evans).² It arises from the coalescence of two separate parts. The larger part develops as an outgrowth from the cranial neural crest; the smaller part is formed by an ingrowing thickening of the ectoderm overlying the mid-brain region. This latter becomes isolated, and after migrating inwards fuses with the former. Nerve-cells become differentiated, and their processes reach the surface of the cell-mass by the shortest route, and spread over its surface, coursing either towards the brain or towards the periphery. Whitehead,³ in a study of the Gasserian ganglion in human embryos, has recently shown that the development of the ophthalmic moiety is always in advance of that going to form the other two divisions. The cells become arranged into clusters sooner, and produce their processes earlier. The centripetal fibres arising from these processes occupy the medial third of what becomes the posterior or sensory root. The cell-mass becomes lobulated so that at the seventh week the three divisions are partially separated. As time advances this subdivision becomes less conspicuous, and by the twenty-eighth week the three parts are imperceptibly blended one with another.

II. ANATOMY.

The Gasserian ganglion is the homologue of the posterior root ganglia of the spinal nerves. It occupies a shallow depression on the upper surface of the petrous bone. It is crescentic in shape, the convexity being forwards, and the two surfaces being directed upwards, forwards, and outwards, and downwards, backwards, and inwards, respectively. The three divisions join the anterior border, and the sensory root emerges from the posterior concave border to drop down into the posterior fossa and enter the pons. The motor root lies at first medial to the sensory root, but soon passes forwards and outwards beneath the sensory root and ganglion to reach the mandibular division. Both motor and sensory roots lie in the same sheath of dura mater. This widens out in front to form Meckel's cave.

R. D. Lockhart⁴ describes the dural arrangement in the following words: "The dura mater of the posterior aspect of the petrous bone near the apex is prolonged forwards like a glove investing the roots, ganglion, and divisions of the Vth nerve, to lie upon the upper surface of the apex of the petrous bone and the sphenoid's great wing near the pituitary 'door-step', and to extend

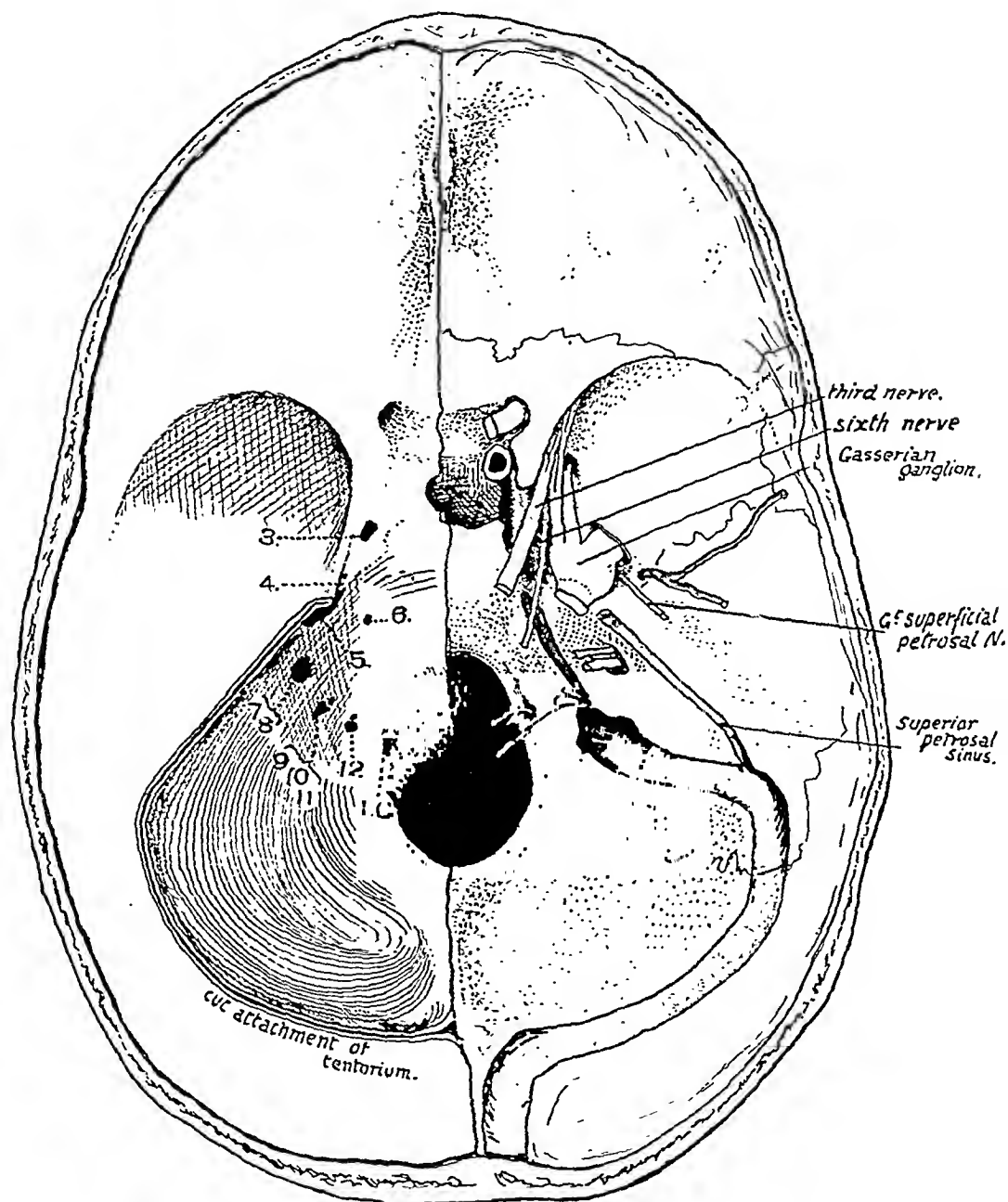


FIG. 75.—Anatomical relations of Gasserian ganglion. (From Frazer's 'Anatomy of the Human Skeleton' (J. & A. Churchill) by kind permission.)

upwards fully half the height of the cavernous sinus, the whole extent of the glove, or cave of Meckel, being concealed by the dura of the middle fossa. . . . the cave is an evagination of the dura of the posterior fossa under that of the middle fossa." It carries with it a prolongation of arachnoid which becomes partially adherent to the anterior part of the ganglion, and the

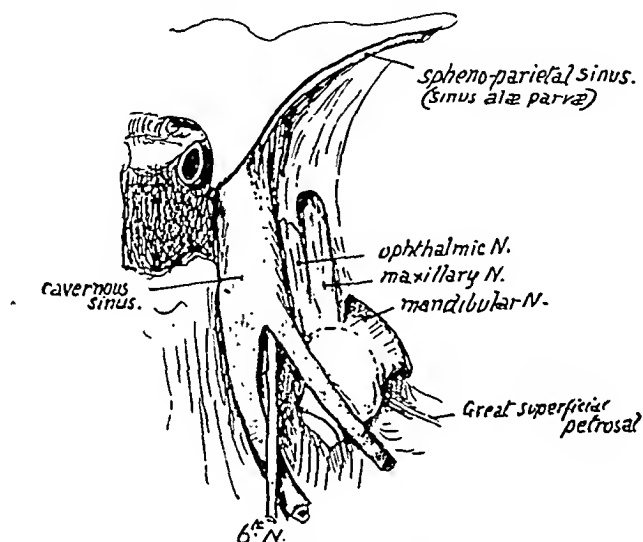


FIG. 76. —Anatomical relations of Gasserian ganglion. (From Frazer's 'Anatomy of the Human Skeleton' (J. & A. Churchill) by kind permission.)

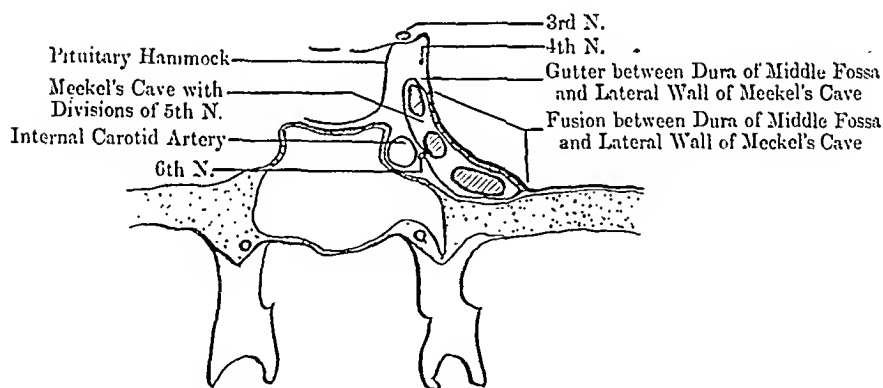


FIG. 77. —Dural relations of Vth nerve. (After Lockhart). By kind permission of the 'Journal of Anatomy'.

ganglion is bathed in cerebrospinal fluid. The great superficial petrosal nerve also lies beneath the ganglion as it passes forwards from the hiatus Fallopii to the foramen lacerum medium, there to be joined by the deep petrosal nerve from the carotid sympathetic plexus, thus forming the Vidian nerve. Unlike the motor root of the Vth nerve, it is extradural throughout its course. Medial to the ganglion is the cavernous sinus, within which lie the IIIrd,

IVth. and VIth cranial nerves and the internal carotid artery; and above these is the pituitary fossa. These points are brought out clearly in *Figs. 75-77*.

The VIth nerve is especially intimately associated with the Gasserian ganglion as it passes from the basisphenoid forwards through a fibro-osseous canal (Dorello) formed by ligaments passing between the base of the posterior clinoid process internally and the apex of the petrous bone externally, and thence into the cavernous sinus. The close relation of the VIth nerve to the apex of the petrous bone is of significance in Gradenigo's²¹ syndrome, to which reference will be made later. Behind and above the ganglion is the superior petrosal sinus. This is easily injured during operations in this locality. Just behind and below the ganglion, i.e., in the posterior fossa, is the internal auditory meatus, through which pass the VIIth and VIIIth nerves; and still farther behind, yet not far removed, are the IXth, Xth, and XIth nerves, converging towards the jugular foramen.

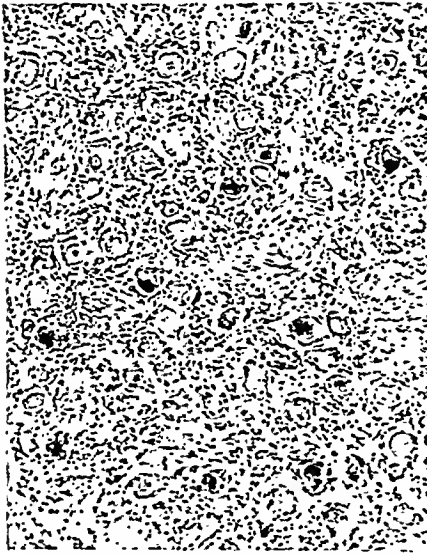


FIG. 78.—Hematoxylin-eosin preparation of a normal Gasserian ganglion. ($\times 65$.)

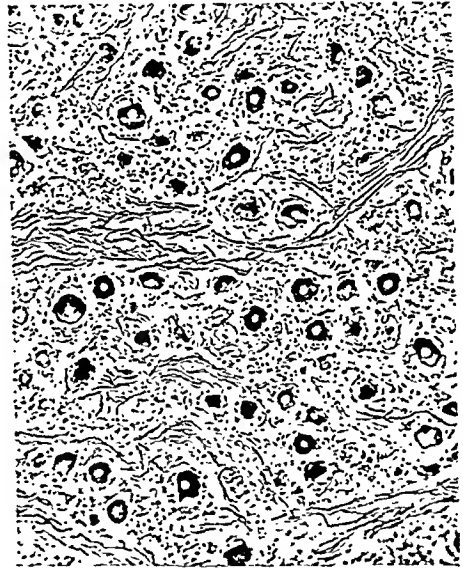


FIG. 79.—Orlandi preparation of the same ganglion showing nerve axons in between the cells. ($\times 65$.)

It can therefore be appreciated how a tumour arising from the Gasserian ganglion may soon produce pressure on near-by structures, causing localizing signs and symptoms. In like manner, a cerebellopontine-angle tumour, an acoustic-nerve tumour, any middle-fossa tumour, or even a tumour of the pituitary body may come to press on some part of the VIth nerve.

As in the case of the posterior root ganglia the ganglion cells, which developmentally are bipolar, become unipolar by fusion of the two processes. *Figs. 78, 79* show many large ganglion cells, each lying within a capsule of flattened cells, epiblastic in origin and homologous with the sheath cells of Schwann. The cytoplasm is finely granular in *Fig. 78*, and the more deeply

stained nucleus contains a well-marked nucleolus. In between the ganglion cells are thin-walled blood-vessels and fine connective tissue, and running in various directions are well-formed medullated nerve-fibres in bundle formation. There are no neuroglial cells. The blood-vessels for the most part have walls of endothelial cells only, though the larger ones are strengthened by an accession of fibrous tissue. *Fig. 79* shows the axons very plainly.

New growths may arise from the ganglion cells, the blood-vessels, the endothelial cells of the vessels, the nerve-fibres or their sheaths, the connective tissue, or finally from the cells forming the capsules of the ganglion cells. The dural sheath of the ganglion may also be the source of a neoplasm, as in one of the patients of the present series, and in many of the cases reported in the literature.

III. THE TUMOUR CASES, WITH A DISCUSSION OF THE LITERATURE.

Tumours of the Gasserian ganglion occur in animals as well as in man (Henschen and Stenstrom⁶). They are either intrinsic or extrinsic—that is, they originate either from the ganglion itself, or from some near-by structure affecting the ganglion by pressure. A number of the cases described under the title ‘tumour of the Gasserian ganglion’ are in actual fact of the extrinsic type, as a study of the text soon shows. Three of the present series are of such a nature.

The first case to appear in literature is that reported by Fischer⁷ in 1863. The first case treated by surgery appears to be that of Krogius⁸ which he reported in 1896. A woman of 37 years of age began three years before with severe pain in the left side of the face. This was soon followed by numbness, weakness of the muscles of mastication, and ptosis, and a little later by a Vth-nerve palsy—all on the left side. The middle fossa was explored and a tumour as large as a pigeon’s egg growing from the sheath of the ganglion was removed. It proved to be an endothelioma. The patient died from meningitis on the twelfth day after operation. Autopsy showed that the tumour was spreading forwards, inwards, and backwards, and the base of the skull in the region of the foramen ovale was so eroded that a portion of the tumour was in contact with the pharynx.

In 1900 Dereum, Keen, and Spiller⁹ reported a case of a man of 32 years of age, of nervous temperament, who had been ailing in health for two years. He had developed a swelling in the left side of the neck, became fatigued easily, and was losing weight. Recently he had suffered pain in the region of the left temple and left side of the vertex. The pain was continuous, and the area involved, which corresponded to the first division of the Vth nerve, was hyperæsthetic. This included the cornea. An enlarged gland was removed from the neck for diagnostic purposes and was reported to be endothelioma. By this time the whole of the trigeminal area was involved, and the masticatory muscles of the left side became weak. The infra-orbital nerve was avulsed, but without relief. It was decided to explore the Gasserian ganglion. A large tumour, apparently arising from the ganglion, was partially excised together with the ganglion itself. No relief ensued, and

neither was there any resulting anæsthesia. A Vth-nerve palsy developed. The tumour was reported as endothelioma. At a second operation a month later every particle of visible tumour was excised. The pain continued unabated, and there still was no anæsthesia; indeed, the whole of the left side of the face remained hyperæsthetic. Mitchell, Ramon y Cajal, and Turner¹⁰ have also reported cases of retained sensation after extirpation of parts of the Vth nerve, and they suggest that probably the VIIth nerve contained sensory fibres in these patients. It is true that a type of subjective deep sensation is conveyed through the VIIth nerve, but in the light of modern experience the maintenance of superficial sensation is suspicious of incomplete section of the ganglion or sensory root.

In 1908 Giani¹¹ published a case of his own, and analysed twenty cases reported in literature up to that time. Of these twenty cases, two were gummata. Only three of the twenty came to operation, two of these being the two already mentioned above. His own case was of a man suffering from continuous pain in the Vth-nerve area, tingling in the same area, paralysis of the motor branch of the Vth nerve of the same side, loss of taste, smell, and visual acuity, tinnitus, deafness, facial palsy, and hemiatrophy of the tongue—all on the same side. Here, then, was evidence of a very extensive lesion involving the Ist, IInd, IIId, Vth, VIIth, VIIIth, and XIIth nerves, but in view of the order of onset of the symptoms, Giani decided to explore the region of the ganglion. He found what appeared to be an endothelioma of the dura overlying and pressing on the ganglion. As much as possible of it was removed, but the patient died of hæmorrhage the next day. Autopsy showed that the ganglion and its branches were flattened beyond recognition, the surrounding bone was eroded, and the cavernous sinus was invaded. The tumour had spread back into the posterior fossa, pressing on the VIIth, VIIIth, and XIIth nerves. The microscopic appearances were of especial interest in that there was the double condition of endothelioma and tubercle. Giani suggests that the primary lesion was probably tuberculous meningitis following on a tuberculous otitis media (the patient suffered from ear trouble during childhood), superimposed on which was the malignant condition initiated by chronic irritation.

Sachs¹² reported a case in 1917 of a woman who began with pains in the area of the first division of the Vth nerve which rapidly spread to the second and third divisions. An impacted molar was extracted, and the paranasal sinuses were drained, but no benefit resulted. Nine months after the commencement of the first pain she developed a paralysis of the external rectus muscle on the same side, and also of the muscles of mastication. The Wassermann was negative. At operation a smooth endothelioma was removed from the region of the ganglion. For six weeks the pain was completely relieved, but after this interval a return of symptoms occurred. The middle fossa was explored for a second time and an inoperable recurrence was revealed. She died eight months later with metastases in the glands of the neck.

Frazier¹³ published two cases in 1918. The first was of an endothelioma of the sheath of the ganglion. It was removed, and complete relief followed. The symptoms in this patient had been pain in the second-division area of the Vth, followed by numbness of the upper lip. The second case was of an

endothelioma which had completely obliterated the ganglion. The symptoms in their order of onset were paralysis of the masticatory muscles, continuous pain in the face, inequality of the pupils, and paralysis of the external rectus muscle—all on the same side. The tumour was removed, but the patient died in two weeks' time.

Gjertz and Hellerström¹⁴ added another case to literature in 1925. A woman, 52 years of age, gave a two years' history of twitchings, tingling, and continuous pain in the right side of the face. On examination there was found to be definite hypo-aesthesia of the cornea, facial weakness, and deafness on the right side, though at this time the muscles of mastication were intact. Nausea and vomiting developed, and very soon afterwards the pain grew much worse, the right masticatory muscles became weak, and ataxia, nystagmus, and inco-ordination of the right hand ensued. At the same time weakness of the soft palate and pharynx on the right side, and of the tongue on the left, became manifest. Here, then, was evidence of involvement of the Vth, VIth, VIIth, and IXth nerves of one side, and of the XIIth nerve of the other. At this stage of the malady a diagnosis of acoustic-nerve tumour was made. Soon afterwards she became incontinent. On the left side she developed a Babinski's sign, an ankle-clonus, and an exaggerated knee-jerk, and the left side of the body became partially anaesthetic. At the same time the right external rectus muscle was paralysed and the patient grew irrational. The Wassermann was negative. The posterior fossa was explored, but nothing was discovered. The patient died. Post-mortem examination revealed a tumour replacing the Gasserian ganglion and having exactly the same gross appearance as an acoustic-nerve tumour. The neoplasm spread to the mid-line, and backwards into the posterior fossa, pressing on the cerebellum and distorting the brain-stem. Microscopic examination showed a typical neurofibroma undergoing cystic degeneration.

In 1928 Altmann¹⁵ reported two cases. The first was of a woman who had suffered from headaches since childhood. In 1915 she experienced a temporary disturbance of vision in the right eye accompanied by pain in the right side of the head and face. This cleared up. In 1917 she was troubled with a temporary auditory disturbance. A year later there was a diminution of visual acuity in the right eye again, and she experienced a burning, tingling sensation in the area of the second division of the trigeminal nerve. She gave positive tests for co-ordination, and was ataxic. The left grip was weak. There was optic atrophy on the right side and a choked disc on the left. There was complete nerve deafness on the right side. Clinically, the case was diagnosed as an acoustic-nerve tumour. X-ray pictures showed destruction of the small wing of the sphenoid, tip of the petrous bone, and the posterior clinoid process on the right side. The radiologist reported 'basal tumour'. The Wassermann was negative. Two weeks later she developed a right-sided proptosis. There was a left homonymous hemianopsia. So great were her headaches that a subtemporal decompression was made. Death occurred soon afterwards. At autopsy a tumour arising from the ganglion was found growing up into the temporal lobe (accounting for the homonymous hemianopsia), and backwards into the posterior fossa causing pressure on the medulla. Histological appearances were those of a neurofibroma undergoing fatty and

LESIONS OF THE GASSERIAN GANGLION 133

mucinous degeneration. Altmann's second case was of a cystic tumour of the sensory root of the Vth nerve spreading backwards into the posterior fossa, giving appropriate signs and symptoms. As in the other cases, pain and partial anæsthesia in the Vth-nerve area were the earliest complaints.

Two cases of endothelioma of the ganglion have recently been published by Dr. Ethel Russell,¹⁶ and C. W. Rand¹⁷ has recorded yet another. Again the sequence of symptoms was pain, paræsthesia or anæsthesia, plus ptosis in one case and weakness of the masticatory muscles in another. All came to operation.

Of the extrinsic tumours, De Martel,¹⁸ in a report on the treatment of cerebral tumours read before the International Society of Surgeons in 1926 in

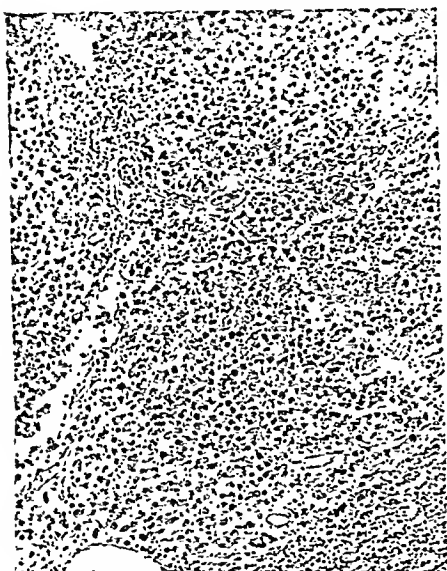


FIG. 80.—Section of an epithelioma of the pituitary which had spread laterally so as to involve the Gasserian ganglion. Chromophil and chromophobe cells are present, the latter being in excess. There are numbers of mitotic figures and a few multinucleated cells. Hæmatoxylin-eosin stain. ($\times 65$.)

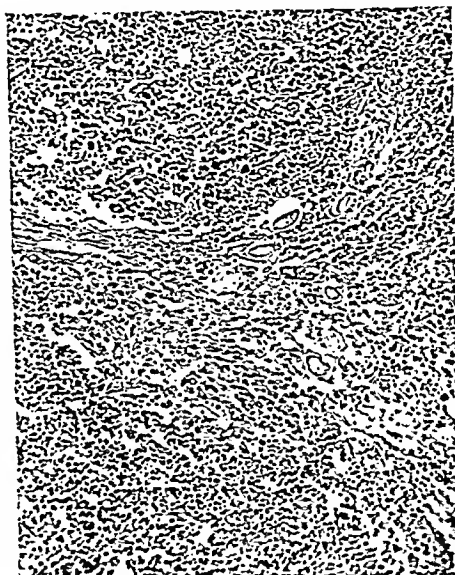


FIG. 81.—Section showing a secondary deposit from a malignant tumour (lymphosarcoma) in the region of the pancreas infiltrating the Gasserian ganglion. Hæmatoxylin-eosin stain. ($\times 65$.)

Rome, cited a case of meningioma of the middle fossa which had grown up into the temporal lobe at the same time pressing on the Gasserian ganglion, giving rise to pain and hypo-æsthesia of the ipsilateral side of the face.

Benson¹⁹ reported a case of a man of 37 complaining of pain and numbness of the face, together with failing vision and anosmia, and a progressive facial weakness—all on the right side. The patient died without any surgical interference. At autopsy a spherical tumour, growing apparently from the region of the tip of the petrous bone, was found. The microscopical report was "epithelial alveolar tumour resembling pars intermedia of the pituitary".

Recently a youth of 20 came to the Mayo Clinic complaining of failing

vision of several months' duration followed by numbness of the right side of the face, and diplopia. There was a paralysis of all of the extrinsic muscles of the right eye, and examination of the visual fields showed a bitemporal hemianopsia. The discs were pale. The pituitary region was explored and an enormous pituitary tumour was found. It was not possible to remove it. It had 'overflowed', so to speak, on to the Gasserian ganglion and ocular nerves. A microphotograph of the tumour (*Fig. 80*) shows epithelioma.

Through the kindness of Dr. Kernohan, Neuropathologist at the Mayo Clinic, I present a section (*Fig. 81*) of a Gasserian ganglion containing a metastatic deposit from a malignant retroperitoneal tumour in the region of the pancreas. The patient was so ill and cachectic from general dissemination of the growth that the specific signs and symptoms resulting from this deposit were masked.

CASES OF THE PRESENT SERIES.

The first four cases here presented are the ones already reported by Dr. Shelden.¹

Case 1.—J. F., male, age 28. Came to the Clinic in February, 1915, complaining of constant pain, burning sensation, and numbness in the right side of the head, face, and tongue.



FIG. 82.—*Case 1.* Section showing masses of vesicular-looking cells with small, very deeply staining nuclei. They appear more like masses of epithelial cells than anything else. Everywhere there are engorged, thin-walled vessels. No ganglion cells could be found, and nerve-fibres are sparse. Hematoxylin-eosin stain. ($\times 65$.)

HISTORY.—There was no family history of any consequence. The right knee-joint was ankylosed as a result of tuberculous disease at the age of 14. Ten years ago he had an abscess of the rectum. At the age of 19 he had typhoid fever, and four years ago suffered from otitis media of the right ear. For some months the glands on both sides of the neck had been enlarged, and three weeks before coming to the Clinic a cold abscess of the left side was incised.

The pain in the right side of the face and head came on several months ago. It was constant, and accompanied by attacks of sharper pain brought on particularly by blowing the nose through the right nostril. Soon afterwards he experienced a burning sensation in the right upper lip and in the right half of the tongue. This was followed by numbness in the same areas five weeks ago. His mouth felt stiff on waking in the mornings, and four weeks ago he noticed that the right pupil was larger than the left one. He slept badly and had a poor appetite. He had bilateral tuberculous cervical glands, and a deflected septum with a very high spur.

ON EXAMINATION.—The patient showed a slight ptosis on the right, and there was a weakness of the right external

rectus muscle. The right pupil was smaller than the left, but reacted to light and accommodation normally. There was no anaesthesia of the cornea. There was

LESIONS OF THE GASSERIAN GANGLION 135

definite hypo-æsthesia of the third-division area of the right trigeminal nerve, including the tongue. The muscles of mastication on the right side were weak. Inspection of the right drum revealed an old perforation. There were no other neurological findings. The Wassermann reaction was negative. The X-ray pictures showed erosion of the posterior clinoid process on the right side.

The administration of iodides by his own doctor had relieved the pain somewhat, so it was decided to continue the iodides and to keep him under observation.

In May he returned. There had been no improvement in his condition. A submucous resection of the nasal septum was performed, but without benefit. In June he developed diplopia, and in July it was found that there was weakness of the right side of the soft palate and anæsthesia of the right cornea. The pain had increased in severity. The second and third divisions of the right trigeminal nerve were at this stage injected, but again no relief followed. It was therefore decided to explore the middle fossa, as the onset of symptoms pointed to an early involvement of the Gasserian ganglion, though there was now evidence of a spread backwards into the posterior fossa producing pressure on the IXth nerve.

OPERATION.—A spherical tumour, half an inch in diameter, was found projecting up from the ganglion. As much of the tumour as possible was removed, and for two weeks the patient was very much relieved. However, the pain then returned with all its old severity. After this he disappeared and it was learned that he died a year later. A section from this case is shown in *Fig. 82*.

Case 2.—H. M. B., male, age 28. First came to the Clinic in November, 1917, complaining of failing vision, and of pain, numbness, and a burning sensation in the right cheek and tongue.

HISTORY.—There was nothing relevant in the family history. He had always suffered from weak eyesight and at no time since birth had he been able to see print with the left eye. A series of operations for strabismus had been carried out on both sides. In 1913 his own doctor made the following report on the state of his vision: "Optic neuritis of the right eye. Optic atrophy of the left eye. Can only count fingers with the left eye. Concentric contraction for white and colours in both fields." Eighteen months ago he began to have "giddy turns accompanied by twitchings of all four limbs", though it was said that he never lost consciousness and never had fits. At this time numbness, pain, and a burning sensation were experienced in the right cheek and tongue. His general condition was good.

ON EXAMINATION.—The right side of the face appeared flatter than the left. The muscles of mastication were definitely weak, so that when the jaw was protruded it moved over to the right. There was hypo-æsthesia of the right cheek and a loss of taste on the right side of the tongue. The pupils were dilated, but both reacted to light and accommodation. Both discs showed secondary optic atrophy. He could only distinguish the outlines of windows with the left eye and fingers with the right eye. The cerebrospinal fluid was under pressure, but normal from a chemical and cytological point of view. The Wassermann reaction of both blood and cerebrospinal fluid was negative. X-ray pictures showed evidence of increased intracranial pressure and expansion of the sella turcica. Labyrinthine tests gave normal though sluggish reactions. There were no other abnormal findings. A diagnosis of tumour of the Gasserian ganglion was made.

OPERATION.—A tumour of the temporal lobe was found pressing down on the ganglion. Some of it was removed. An uninterrupted convalescence followed.

SUBSEQUENT PROGRESS.—Immediately subsequent to operation the pain was much relieved, but there was no improvement as regards the other symptoms, and some œdema of the discs developed. He gradually became completely blind, and in 1924 had to be removed to a State Hospital.

The sections of the preserved specimen show only normal brain tissue with many small hæmorrhages into it. The pathological report at the time of operation was 'glioma'. Judging from the long pre-operative and post-operative history this was presumably of the astrocytoma type (Cushing and Bailey).

Case 3.—J. Z., a married woman, age 47. Came to the Clinic in May, 1918, complaining of increasingly severe continuous pain in the left side of the head and face, and a tingling sensation in the same area.

HISTORY.—The family history was of no consequence. The pain commenced eleven months ago. She had some teeth extracted and the paranasal sinuses were drained, but no improvement resulted. The pain was becoming worse, and superimposed on its continuous nature were sharp attacks of stabbing pain brought on especially by exposure to cold and wet. Six months ago she experienced double vision, and noticed that she was going deaf in the left ear. Her general condition was good.

ON EXAMINATION.—The left pupil was seen to be dilated and fixed, and there was almost complete ophthalmoplegia of the left eye. The fields were normal: the left disc was slightly blurred. There was hypo-aesthesia of the left temple and left side of the face. The muscles of mastication on the left side were distinctly weak, and there was left-sided nerve deafness. The left half of the tongue was atrophied. The Wassermann reaction of both blood and cerebrospinal fluid was negative. The spinal fluid was not under pressure, and there was nothing abnormal chemically or cytologically. No other abnormality was found.



FIG. 83.—Case 3. This is very similar to Case 1. There are masses of epithelial-like cells. There are many thin-walled blood-vessels. In this section there are ganglion cells which present various forms of degeneration, such as retraction from their capsules, pigmentation, eccentricity of nuclei, and poor staining. In view of the close relation of the cell-masses to the vessels, this tumour was originally reported as a perivascular angiosarcoma. Haematoxylin-eosin stain. ($\times 65$.)

ataxic, exhibiting Romberg's sign. There was a mass of glands in the left side of the neck. Here, then, was evidence of extensive spread in the left posterior fossa, with newly developed signs of pressure on the facial and vagus nerves, and on the cerebellum. The glands were suspicious of metastatic deposits, though neither biopsy nor necropsy was performed. No treatment was given, and the patient died the following year. A section from this case is shown in *Fig. 83*.

Case 4.—J. M. D., a married woman, age 45. Came up to the Clinic in May, 1918, complaining of headache, pain in the left side of the face, hoarseness, and difficulty in swallowing.

HISTORY.—There was no personal or family history of consequence. Two years ago she received a blow on the forehead, but apparently suffered no ill effects.

A diagnosis was made of basal tumour arising in the region of the ganglion and spreading back into the posterior fossa pressing on the IIIrd, IVth, Vth, VIth, VIIth, and XIIth nerves.

OPERATION.—It was found that the ganglion was destroyed by a large bluish-red granular tumour. As much of the tumour as possible was removed, and the sensory root of the Vth nerve was cut. Convalescence was uneventful, and she was relieved of her pain.

SUBSEQUENT PROGRESS.—The patient was seen again in December, 1919, when she stated that until a few weeks before she had remained free from her old pain. Together with its return she had developed huskiness of the voice, a cough, left facial paralysis, and complete blindness of the left eye. She also developed pain and weakness in the left arm. On examination it was found that the left vocal cord was paralysed. The patient was

A year and a half ago she began to experience severe and continuous pain and headache on the left side of the head and face. This was thought to be due to antrum trouble, and a doctor drained her antrum through the mouth. She had no relief. Later on she had alcohol injections, and though the face became numb the pain was in no way alleviated. She also had her teeth extracted. More recently she has become increasingly deaf, especially in the left ear. Two months ago she lost her voice, and has been hoarse ever since. During the same time she has had difficulty in swallowing, the food tending to return down the nose. She had several short convulsions accompanied by vomiting.

ON EXAMINATION.—The left pupil was smaller than the right, and reacted poorly to light. There was a slight ptosis on the left side; the corneal reflex was sluggish; and there was a paralysis of the left external rectus muscle. The fields and fundi were normal. There was diminished sensation in the Vth-nerve area on the left side, and the muscles of mastication were weak on the same side. There was no facial paralysis, but a lack of tone in the facial muscles on the left side. Hearing was markedly diminished on both sides, tests showing nerve deafness on the left. Caloric tests gave a negative response on the left. There was weakness of the left palate and pharynx. The left side of the tongue was paralysed, so too was the left vocal cord. The abdominal reflexes were absent. The knee- and Achilles-jerks were exaggerated on both sides, and there was a Babinski's sign on both sides. The patient had lost 20 lb. in weight.

A diagnosis of basal tumour was made, and in view of the fact that pain in the trigeminal area was an early symptom, it was decided to explore the middle fossa in the region of the Gasserian ganglion, in the hope that it would be possible to avulse the sensory root in order to relieve some of the pain.

OPERATION.—The dura was found to be so adherent to the ganglion and tumour which involved it that it was opened. As much as possible of the tumour was removed, though it was realized that it extended into the posterior fossa and involved the ocular nerves.

SUBSEQUENT PROGRESS.—After the operation the patient developed a left facial paralysis which was probably due to swelling in the posterior fossa. Otherwise she recovered from the operation well. However, she died in December of the same year. The tumour was reported at the time to be malignant, so the early death was not surprising in view of the fact that already at the time of the operation there were signs of involvement of almost all the cranial nerves on the left side, and of pressure on the pyramids too. A section from this case is shown in Fig. 84.

Case 5.—S. I. T., a married woman, age 52. Came up to the Clinic in October, 1923, complaining of pain and tingling in the right side of the face and tongue.

HISTORY.—There was no relevant family history. Apart from typhoid fever at the age of 14 years she had suffered from no previous illnesses. Four years ago she had experienced a "creeping, tingling sensation" in the right infra-orbital area and buzzing noises in the head. There had also been sharp, shooting pain in



FIG. 84.—Case 4. Section showing whorls of fibrosarcomatous cells invading the ganglion. The darkly staining structure in the middle may or may not be a degenerated ganglion cell. Hematoxylin-eosin stain. ($\times 65$.)

the right temporal region which was brought on by touching the infra-orbital region as in washing or powdering the face. These complaints lasted six months and then subsided. Eight months before coming to the Clinic the same pain and paræsthesia returned. The act of eating sometimes brought on acute pain in the region of the right temporo-mandibular joint and temporal fossa. During the last five months the pain had become paroxysmal, and on occasions was so severe as to make the patient cry out. She was taken to a local hospital, where the infra-orbital nerve was avulsed. No immediate relief followed, though the pain gradually became less severe and lost its paroxysmal character. Two months ago the frontal sinuses were drained, and one month ago the tonsils were removed. Quite recently she noticed a tingling sensation in the right side of the tongue.

ON EXAMINATION.—There was found to be definite impairment of sensation over the upper lip and the side of the nose on the right side. A thorough neurological investigation revealed nothing else abnormal, and a diagnosis of chronic nervous exhaustion was made.

A year later she returned to the Clinic. In the meantime she had taken a course of baths, massage, and treatment for intestinal toxæmia elsewhere, and considered that she had derived benefit from these measures. However, two months ago she noticed a burning sensation and numbness of the right side of the tongue. This subsided for a little while, only to return. A week ago on rising in the morning she had a sudden attack of extremely severe pain in the upper part of the right side of the face. This was followed by a series of typical *tie douloureux* attacks so bad as to make her fearful of attempting to eat. Apart from anæsthesia of the second-division area of the Vth nerve, findings were essentially negative.

OPERATION.—A red granular mass was found growing from the dura over the Gasserian ganglion and pressing on it and on the sensory root, rendering them red and congested. The tumour was removed. Apart from the development of a right facial paralysis which soon cleared up, the convalescence was without complication, and the patient was discharged from hospital three and a half weeks after the operation.

SUBSEQUENT PROGRESS.—The patient was seen again one month after discharge. There was still a slight weakness of the right facial movements, and the muscles of mastication on the right side were weak. The cornea was anæsthetic, and she said that she felt as though there was 'running water' in her right ear. She still had a little pain, but was so relieved that she was well pleased that she had undergone the operation.

No specimen was available for study in this case, but according to the operation notes the tumour must have been a meningioma.

Case 6.—A. W., a married woman, age 48. Came up to the Clinic in January, 1928, complaining of pain and numbness in the left temple, upper jaw, and cheek, and also drooping of the left eyelid.

HISTORY.—There was no family or personal history of consequence. The pain had come on about two years ago. Eight crowned teeth were extracted, and she was relieved from the pain for about three months. Then the pain became continuous, and she experienced a drawing sensation in the left cheek and a feeling of fullness in the left ear. Three months ago her left antrum was drained, and though she had had no pain since, the numbness, ptosis, and fullness of the ear had continued. Recently she had experienced diplopia from time to time.

ON EXAMINATION.—The left pupil was dilated and fixed, and there was marked ptosis. The left external rectus muscle was paralysed, and there was some weakness of the other extrinsic muscles of the left eye. The fields and fundi were normal. There was anæsthesia of the left cornea. The left angle of the mouth seemed to droop a little, and the muscles of mastication on the left side were markedly weak. The left side of the soft palate moved sluggishly when the reflex was elicited. There was diminished sensation in the left side of the forehead and over the left cheek.

Diagnosis lay between meningo-encephalitis and Gasserian ganglion tumour. It was decided to operate.

LESIONS OF THE GASSERIAN GANGLION 139

OPERATION.—A large tumour arising from the ganglion was found and removed. The convalescence was uneventful.

SUBSEQUENT PROGRESS.—This patient was seen again in August, 1928. She was then in very good health. There was no pain, though the tingling and drawing sensation in the left cheek still affected her. However, this was much less severe than before operation, and did not really worry her very much. There was complete anæsthesia of the whole of the left Vth-nerve area on the left side. The muscles of mastication appeared stronger, and the angle of the mouth was not so drooping. The left pupil was still fixed and dilated, there was a complete Vth-nerve palsy, and a partial IIIrd-nerve palsy. The visual acuity of the left eye was 6/60; that of the right eye was 6/6. A section from this case is shown in Fig. 85.

Case 7.*—A. G. C., a single woman, age 29, a school teacher in a convent. Came to the Clinic in May, 1929, complaining of pain and numbness of the left side of the face, left-sided facial paralysis, and ulceration of the left cornea.

HISTORY.—There was nothing relevant in the family or personal history. The trouble commenced six years ago with pain in the left ear and a 'tight feeling' up to the vertex. Then gradually numbness of the left side of the tongue and face developed, added to which were periodic attacks of sharp, shooting pain in the same area. Two years ago she noticed that the left side of the face looked stiff, and following this a facial paralysis rapidly developed. Six months ago she began to develop painless ulcers of the cornea on the left side, and during this time has had about thirty such ulcers. They have been quite small, and have left very little scarring. She says that she sometimes has a tightening sensation down the right arm and leg.

ON EXAMINATION.—There was complete facial paralysis on the left side. There were several small ulcers on the left cornea, the eye was red, and the episcleral vessels were engorged. There was complete corneal anæsthesia. There were no ocular palsies, and the fields and fundi were normal. There was partial anæsthesia of the forehead, cheek, and tongue on the left side. The patient complained of feeling "as though the Eustachian tube were blocked", this being due probably to paralysis of the tensor palati muscle. No other cranial palsies were revealed. There was nothing else abnormal.

In this case there was evidence of some lesion starting in the locality of the ganglion and spreading backwards into the posterior fossa. The nerves most affected were the first and second divisions of the trigeminal, and the VIIIth. There were no abnormal findings to account for the complaint of occasional paræsthesia in the right arm. A diagnosis of Gasserian ganglion tumour was made.



FIG. 85.—Case 6. This section is typical of arachnoid-fibroblastoma, the name applied by Mallory and Penfield to the old 'endothelioma of the dura'. Cushing and Bailey prefer to call it 'meningioma'. It is very vascular. There was one ganglion cell in this section, though not included in the photograph. Hæmatoxylin-eosin stain. ($\times 65$.)

* I am indebted to Dr. J. R. Learmonth for permission to include this case, which is being published from a pathological point of view by him and Dr. Keruohan at a later date.

OPERATION.—On exposing the ganglion a semi-gelatinous mass was seen to arise from it. As much as possible was scraped out. There was no serious bleeding. The wound was closed apart from the usual iodoform wick drain. The convalescence was uninterrupted.

In view of the fact that it was impossible to remove that part which had spread back into the posterior fossa through the middle-fossa approach, the question of a posterior-fossa exploration at a later date was considered, as also was one of the facial-nerve cross-anastomosis. However, it was decided that no useful purpose would be served by either of these measures. The prognosis is of course

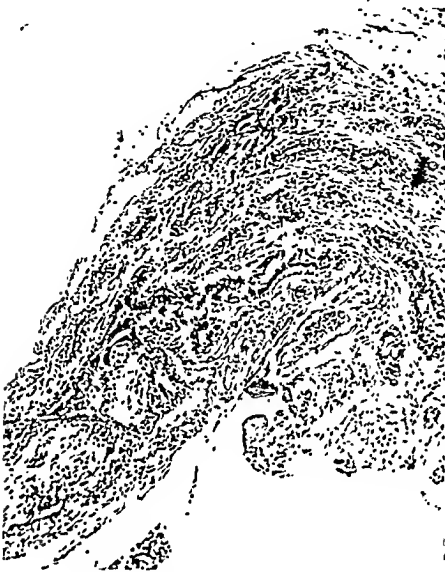


FIG. 86.—H&E stain. ($\times 65$.)



FIG. 87.—Zenker stain. ($\times 135$.)

FIGS. 86, 87.—Sections showing an arrangement of cells different from that in any of the other cases. The ganglion cells are small and shrunken, and some are pigmented. The capsular cells are not well-marked, and indeed in some places seem to be missing. The neoplastic cells are arranged in acinar, columnar, and tubular formation, and in some places there is a suggestion that in the middle of an acinus is a degenerated ganglion cell. May this not be a cytoplasia of the capsular cells?

bad, and examples of similar cases from the literature have already been cited. The patient had no pain during her stay in the hospital, but it is too early to claim that this was due to the operation. Sections from this case are shown in Figs. 86, 87.

IV. THE INFLAMMATORY CASES, WITH A DISCUSSION OF THE LITERATURE.

Inflammatory lesions of the Gasserian ganglion are extremely rare, and I can find mention of only three cases in the literature.

In Giani's paper, which has already been referred to, mention is made of two cases which fall in the inflammatory group. These were two patients who died and upon whom post-mortem examinations were performed. In each case the Gasserian ganglion was found to be the seat of gummatous inflammation. Unfortunately no clinical notes are given. In connection

with this it is of interest that severe pain in the trigeminal area is a comparatively frequent event in luetic patients, though in the cases reviewed there was no suggestion of any anæsthesia or paræsthesia, so presumably there was no lesion producing destruction of any part of the ganglion as in the tumour cases.

Fothergill, in 1783, originally described the paroxysmal type of facial pain, adding that "some painful affections of the head which sometimes spread to the face, arise from ancient venereal complaints imperfectly cured." Nonné²⁰ stated that "the trigeminal is especially often affected in the form of an isolated and simple neuralgia". Mott²¹ recorded a case of trigeminal neuralgia in a syphilitic young woman cured by mercury and iodides. Veits²² reports a case of a man of 33 who for nine years suffered from typical tic douloureux in the second division. The Wassermann was negative. Alcohol injection relieved the condition for six months; then the attacks returned. On his second visit the Wassermann reaction was found to be positive. A course of one of the intravenous arsenic preparations was given, progressive improvement following on each successive injection. After the fourth dose the attacks ceased altogether, and he was still free from pain at the time of publication six months later.

The third case from literature is that of Carrol Smith²³ published in 1925. The patient suffered from chronic otitis media. A sudden flare-up occurred and an inflammatory swelling appeared above and in front of the ear. A mastoidectomy was performed, but little pus was found in the mastoid air-cells, though the swelling in front of the ear contained pus under tension. The lateral sinus and the dura were not exposed. The temperature did not settle, so it was decided to re-open the mastoid wound and to expose the dura and lateral sinus. These were found to be normal. The patient then developed meningitis and complete anæsthesia of the face on the same side as the affected ear. Death supervened. Autopsy showed a generalized meningitis, but there was neither cerebral nor cerebellar abscess. In the position of the ganglion on the anæsthetic side was a white elevated area as big as a florin covered over by thinned-out dura. On incising this about 2 c.c. of thick pus escaped. The abscess entirely replaced the ganglion. No disease of the sinuses or bone could be found.

At this point it would seem appropriate to say a few words about a syndrome first described by Gradenigo²⁴ in 1904. This consists of otitis media with or without involvement of the mastoid, accompanied by Vth-nerve palsy, and sometimes by pain in the trigeminal area. Gradenigo attributed these symptoms to infection of the air-cells at the tip of the petrous part of the temporal bone. This leads to congestion and œdema in the fibro-osseous canal through which the Vth nerve passes, as already emphasized in discussing the anatomy, and also in the region of the ganglion as it lies over these cells. The syndrome is recognized by most authorities, but its interpretation is a matter of controversy. Vogel²⁵ considers it to be due to a localized meningitis over the apex of the petrous bone. Kuiek,²⁶ on the other hand, thinks it due to a toxic neuritis. Regarding the pain, some authorities put forward the theory that it is of a referred nature, the are being the chorda tympani, auriculotemporal, recurrent branch of the mandibular

nerve, or the great superficial petrosal nerve. Carrol Smith denies this hypothesis. Weiner²⁷ reports a case of a woman of 38 who for several years complained of severe neuralgic pains in all three areas of the trifacial. She was suffering from otitis media. Then she developed a ptosis of the same side. Mastoidectomy was followed by complete cure, even to the ptosis. Cases have also been recorded by J. M. Wheeler,²⁸ Perkins,²⁹ Weiner,³⁰ Friedenwald,³¹ Dench,³² and others. In all cases recovery took place, so that it is only possible to speculate as to the process going on in the ganglion. In Dench's case the floor of the middle fossa was actually drained, though no pus was found.

The question of herpes in the Vth-nerve area is as controversial as that of tic douloureux. Occasionally herpes follows section of one of the divisions of the Vth nerve for tic douloureux, and some authors consider this to be due to the cutting of sympathetic fibres which occurs at the same time. Herpes of the face, particularly of the supra-orbital area, may attack a patient just as herpes zoster does in any other part of the body; and Henry Head,³³ in his monumental work on herpes zoster, described cases of facial herpes in which after death years after the attack, lesions were found in the Gasserian ganglion exactly similar to those found in posterior root ganglia of patients suffering from herpes of the trunk—that is, an initial hæmorrhage, followed by small-round-celled infiltration, proliferation of the blood-vessels, and thickening of the pia. This process eventually leads to scar formation. These examples cannot be claimed to be of an inflammatory nature. On the contrary, the herpes of acute infective fevers, and especially that of pneumonia, can only be explained by a passing mild infection and congestion of the ganglion. No permanent damage is done, and these patients never become the victims of post-herpetic neuralgia.

CASES IN THE PRESENT SERIES.

The first of the two cases of the present series is one of tuberculous infiltration of the ganglion. The second one, although having been under observation for six years, is still of doubtful diagnosis.

Case 8.—S. C., female, age 44, school teacher. Came to the Clinic in July, 1924, complaining of pain in the right side of the face and head, and a tingling sensation in the second- and third-division areas. There was also ptosis and diplopia of the right eye.

HISTORY.—Apart from the early headaches described below there was nothing of consequence in her previous history. One brother had died of tuberculosis. During adolescence she had suffered from periodic occipital headaches. These ceased during her early twenties, but commenced again two years ago, but with this difference, that they were now generalized. There had been no nausea or vomiting. Four months ago she noticed an itching sensation inside the right nostril, followed by numbness of the same area and of the outside of the nose also. Soon after this she developed very severe pain in the right cheek which within a week came to involve the whole head. She had attributed these complaints to a cold. The right antrum was found to be infected, and was drained. It cleared up well, and for a short time the pain was less. However, it returned as a continuous dull ache, superimposed on which were intermittent attacks of very severe pain involving the whole of the right side of the face. The itching and numbness of the nose also became worse, and two weeks ago the right eyelid was noticed to droop. One week ago

she developed diplopia, and for the three days prior to coming to the Clinic she felt nauseated.

ON EXAMINATION.—The pupils were equal and reactive. There was a weakness of the right external rectus muscle, and a slight degree of ptosis on the same side. There was vertical nystagmus on both sides. The cornea was anæsthetic; the fields and fundi were normal. There was definite diminution of sensation of the nose and forehead up to the hair-line on the right side. The muscles of mastication on the right were weak. The Wassermann reaction was negative, and the cerebro-spinal fluid normal. Nothing else abnormal was discovered, and a diagnosis of Gasserian ganglion tumour was made.

OPERATION.—On exposure the ganglion and its three divisions appeared as though the seat of a neurofibroma, and were about three times normal size. The whole ganglion was removed. One of the ocular nerves was exposed and thought to be traumatized during dissection. Convalescence was uninterrupted.



FIG. 88.—*Case 8.* This section presents what must be a unique picture. To the one side are fairly normal-looking ganglion cells, nerve-fibres in bundles, and blood-vessels. To the other side are definite tubercles. None has gone to caseation. Most show multinucleated giant cells surrounded by epithelioid cells, these in turn being surrounded by small round cells. In between the healthy and diseased parts degenerated ganglion cells are intermingled with tubercles. Some of these latter are so young as merely to be represented by a few epithelioid cells surrounded by a few lymphocytes, no central multinucleated giant cell being present. *Hæmatoxylin-eosin stain. (× 65.)*

SUBSEQUENT PROGRESS.—The pain absolutely disappeared. The movements of the external rectus muscle began to improve, and at the end of 1927 the patient wrote to say that she was doing her ordinary work quite comfortably. Only occasionally did she experience diplopia. A section from this case is shown in Fig. 88.

This case is different from Giani's patient, in whom both endothelioma and tubercle were present. As already stated, in his case the ganglion was crushed beyond recognition so that it is not known whether there was any tuberculous infiltration of the ganglion. The demonstrable lesion was in the dura lying over the ganglion. Giani thought this tuberculous condition was the result of a localized tuberculous meningitis spreading in from a tuberculous otitis media of childhood. Leonard G. Parsons has pointed out that tuberculous infection of the middle ear is not an uncommon condition in young children, and may pass by unrecognized. *Case 8* had no history of ear trouble.

and the lesion found at operation affected the ganglion only. She did, however, suffer from headaches during adolescence. It is impossible to say whether or not there was any connection between these two conditions, but a comparison of these two cases is of interest.

Case 9.—T. N. F., a married woman, age 27. Came to the Clinic in July, 1923, complaining of pain in the right side of the head and face, and numbness in the right side of the face.

HISTORY.—There was nothing in the family history or her own previous history of consequence. The trouble commenced twelve weeks ago as a sharp shooting pain over the right eye. A few weeks later she began to have attacks of pain which extended up to the top of the head and lasted about two hours. Her scalp became sore so that it hurt her to comb her hair. During the last ten weeks she had not been free from pain during any one whole day. Five weeks ago she went into hospital elsewhere to have her paranasal sinuses drained. This procedure afforded temporary relief, but the discomfort soon returned. At this time it was brought on by eating or talking. Three weeks ago three teeth were extracted, and following this she noticed a numbness of the right side of the face. She thought that her vision was affected during the attacks. She had never been nauseated and had never had fits. She had been taking large amounts of aspirin and strong tea. She looked in good health, but was very nervous about herself, fearing that her discomfort signified impending paralysis. Indeed, there was a big hysterical element.

ON EXAMINATION.—It was observed that the right pupil was larger than the left, but reacted to light and accommodation normally. The right external rectus muscle was weak, as also were the muscles of mastication on the right side. The fields and fundi were normal. There was a definite diminution of sensation of the right side of the face, particularly of the first-division area, and the cornea was completely anæsthetic. She was slightly deaf in the right ear. Reflexes in general were somewhat brisk, but there was none of abnormal type. There was anæsthesia of the pharynx. Nothing else abnormal was detected. The Wassermann was negative.

A diagnosis was made of middle-fossa tumour, probably of malignant nature, and pressing on the Gasserian ganglion, and operation was advised.

OPERATION.—A tumour of the ganglion was found and removed. The posterior root was avulsed: 50 mgrm. of radium were inserted for twenty hours.

SUBSEQUENT PROGRESS.—Convalescence was without any untoward incident. The pupils became equal, and the external rectus regained its power.

The patient returned in February, 1924, for review. The right pupil was again larger than the left, and the masticatory muscles on the right side were completely paralysed. There had been no progression of the disease. Her tonsils were removed, and a course of radium was instituted.

In June of the next year she was again examined, and the examination was essentially negative. She still complained of various discomforts, especially in the posterior part of the parietal region. The small occipital nerve was blocked with novocain. In September she returned, giving an account of two attacks of what sounded like *petit mal*. She said that she became 'dazed' from time to time, suffered from headaches, and vomited occasionally. Her deafness had become worse. A year later she gave a story of a continuation of the *petit mal* attacks. The fundi were normal, but there was a left homonymous hemianopsia for colours.

In September, 1927, it was found that there was some concentric contraction of the fields, but that the homonymous hemianopsia had disappeared. She still complained of headaches and discomfort in the right side of the head and face. A further twelve months later she came up to the Clinic again. On this occasion she stated that during the preceding twelve months she had felt much better, but that two weeks previously she had developed symptoms on the left side exactly similar to those originally on the right side. On examination the signs were exactly as they had been on the right side formerly. Tests for co-ordination gave positive

results equally on both sides. There was a little ataxia, and Romberg's sign was present. She declared that she had 'triple vision'. In view of the inflammatory nature of the condition on the right side, it was thought that this new train of symptoms was probably due to a similar condition on the left side. A course of X-ray and radium was therefore administered, and she obtained definite improvement thereby.

In March, 1929, she was re-examined. She claimed that her visual acuity in the right eye was becoming progressively worse. The fields showed a relative central scotoma on the right side, but again there was no homonymous hemianopsia. She still had the petit mal attacks.

I saw this patient myself in April, 1929. Her condition was the same as the month before. She was unable to carry out any of the co-ordination tests, and exhibited Romberg's sign. That there is a very considerable functional element in this case there is no doubt. Indeed, the patient seems to find great satisfaction



FIG. 89.—Case 9. This section is difficult to interpret. There is a small-round-cell infiltration of the ganglion substance. These cells can be seen between the individual ganglion cells, and also in amongst the nerve-fibres. The ganglion cells show nothing in particular. The picture is that of inflammation of a non-specific type. Hæmatoxylin-eosin stain. ($\times 65$.)

in her inability to carry out various tests. Nevertheless, an inflammatory tumour was removed from the one ganglion, and she must be given credit for this. Her mentality seems clear, and she talks clearly though volubly. Though she complains of deafness, she apparently hears conversation in ordinary tones perfectly well. She looks pale and thin.

I cannot venture a diagnosis. Dr. Parker, who has watched this case for six years, points out that the varying visual fields, at one time showing an homonymous hemianopsia, at another a concentric contraction, and at yet another a relative central scotoma, are suggestive of multiple sclerosis. However, the section of the tumour removed at operation is not the picture of multiple sclerosis (*Fig. 89*).

V. THE PATHOLOGY.

It has been my privilege to discuss the sections of these tumours with Professor Haswell Wilson, Dr. Mallory, and Dr. Penfield, as well as with Dr. Kernohan. The following remarks are based on their combined opinions. The pathology of the seven tumour cases may be summarized as follows:—

Case 1.—Epithelioma. Ganglion tissue present—supported by operation notes and by the presence of nerve-fibres.

Case 2.—Glioma.

Case 3.—Epithelioma. Ganglion cells and nerve-fibres present.

Case 4.—Fibrosarcoma. Ganglion cells and nerve-fibres present.

Case 5.—Meningioma. This is presumed from the operation notes.

Case 6.—Meningioma. One ganglion cell was found, though this is not included in the photograph.

Case 7.—An unclassified tumour which, however, undoubtedly arises from within the ganglion.

In *Case 2* the tumour was extrinsic and affected the ganglion by pressure only. As regards *Case 5*, there was unfortunately no tissue available for study, and no pathological report in the notes. According to the operation notes, however, the ganglion was affected by pressure only, the tumour arising from the dura overlying the ganglion. It was presumably, therefore, a meningioma. The sections in *Cases 3, 4, and 7* all show ganglion cells and nerve-fibres infiltrated by tumour cells. Although no ganglion cells could be found in over one hundred consecutive sections from *Case 1*, nerve-fibres are present. So, in conjunction with the operative notes, this may justifiably be considered to contain ganglion tissue which has been infiltrated by tumour cells and not merely affected by pressure. The individual appearance and general arrangement of the neoplastic cells in *Cases 1* and *3* are exactly similar, and are almost certainly epithelioma. As to the origin of these cells, it is difficult to be certain. Many cases are on record of epithelioma of the upper jaw invading the pterygo-maxillary fossa and then the floor of the middle fossa; but neither of these cases gave any history suggestive of upper-jaw malignancy. Another source would appear to be from that smaller part of the ganglion which develops from ectodermal ingrowth, as described on p. 126.

It is assumed from the operation notes that *Case 5* was an endothelioma (or meningioma), and *Case 6* most certainly was, as shown by the sections. Only one solitary ganglion cell was found in many sections of the latter, and no nerve-fibres could be discovered. It is most probable, therefore, that this tumour was wholly extrinsic, and that the one ganglion cell came from a small nodule of adjacent ganglion.

In the literature many of the so-called 'endotheliomata of the Gasserian ganglion' are alleged to have arisen from the endothelial cells lining the dural capsule of the ganglion. Yet the sections accompanying the description of these cases clearly show definite infiltration of the ganglion tissue by the neoplastic cells. Now, since the ganglion is bathed in cerebrospinal fluid and has a sheath of arachnoid as well as of dura, a meningioma of this sheath bears the same relation to the ganglion as a parasagittal meningioma does to the brain. These latter tumours do not infiltrate the brain unless they become malignant; they merely indent it. There is, then, no reason to suppose that a meningioma of the sheath of the ganglion would infiltrate the ganglion unless it became malignant. I am of the opinion, therefore, that endotheliomata which can be seen actually to infiltrate nerve-fibres and ganglion cells in the Gasserian ganglion have their origin either within the

ganglion—probably from the vascular endothelial cells—or from a meningioma which has become sarcomatous.

The sections from *Case 4* contain tumour cells which in some parts resemble endothelial cells, but in other parts look more like fibrosarcoma cells. The operation notes state that the ganglion tumour was very adherent to the dura, which is in favour of sarcoma. This may have arisen from either the connective tissue in the ganglion or from the dura. The general disposition of the tumour cells in *Case 7* leaves no doubt that this too is a true intrinsic tumour of the ganglion, and at operation the dura appeared unaffected.

Thus we may say that possibly four of these tumours are intrinsic tumours of the Gasserian ganglion.

VI. OPERATIONS.

Associated with the story of the progress in the surgery of the Gasserian ganglion are the names of Rose, Hartley, Krause, Horsley, Macewen, Ballance, Trotter, Cushing, and Frazier. At the present time there is a fair degree of uniformity in the method of approach, namely, through the temporal fossa, and only in smaller details do surgeons differ. Being most familiar with Dr. Adson's technique I will describe his operation in brief.

The patient is placed on the operating table, which, after the patient has been anæsthetized, is inclined to an angle of about 70°. The head is then fixed in a clamp in such a manner as to render the zygoma horizontal, and the coronal plane slightly tilted down to the affected side to allow fluids to run out, thus preventing a collection in the depths of the wound. The incision is a straight line running from a point 4 cm. above the tip of the ear to one 1½ cm. in front of the ear on the upper border of the zygoma, as advocated by McEvoy. The skin, temporal fascia, and temporal muscle are all divided in this line, and the bone of the temporal fossa is exposed. The anterior and posterior leaves of the fascia are incised for about ½ cm. along the attachment to the zygoma to allow of their being retracted forwards and backwards. An adequate amount of bone is removed, the lower margin of the opening being level with the floor of the middle fossa. By means of pieces of moist wool on forceps the dura is gently separated inwards from the floor of the middle fossa until the middle meningeal artery is brought into view as it enters the skull through the foramen spinosum. A blunt hook is used to free it all round, and the vessel is then tied and cut. The separation of the dura is then continued inwards until the third branch of the trigeminal is seen. This exposes what is known as the line of cleavage between the dura of the middle fossa and the dural sheath of the ganglion. This sheath is incised from before backwards, thus bringing into view the pulsating arachnoid. On opening this the sensory root is exposed.

This is the operation as performed in *Cases 2* to *9*. The notes do not record the method of approach in *Case 1*. In *Cases 3, 4, 8, and 9* the sensory root was cut. The accompanying sketch (*Fig. 90*), which was made during the operation in *Case 9*, is included by the kind permission of Dr. Adson.

In treatment of tic douloureux it is from this point of the operation that surgeons vary in details. In days gone by individual branches of the ganglion were avulsed. Then the excision of the whole ganglion became popular, but

this measure was difficult and dangerous where a simpler procedure would suffice. It was Spiller and Trotter who pointed out that the essential step in relieving pain was to cut the posterior root behind the ganglion. Some surgeons cut through the whole of the posterior root, regardless of whether or not the motor root be included. Dr. Adson saves the motor root in all cases of tic, though of course this was not possible in all the tumour and inflammatory cases. He does this by hooking aside the sensory root from above. When the sensory root is thus kinked, the motor root springs into view and so can easily be saved. Frazier cuts only the outer two-thirds of the sensory root and saves the motor root. This is in order to save those fibres going to form the ophthalmic division, thereby preserving corneal sensation and preventing corneal ulceration. The efficacy of this manœuvre is a matter of opinion.

This operation is very safe, and Dr. Adson's mortality in over 500 cases has been just about 1 per cent.

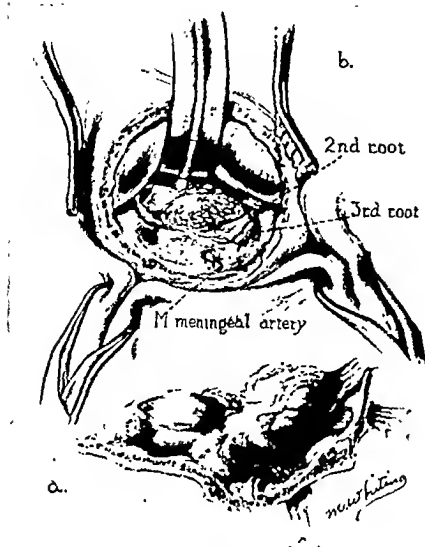


FIG. 90.—b, The ganglion involved by tumour. The middle meningeal artery, and second and third divisions have been divided; a, The condition of the ganglion after exposure but before dissection.

VII. DIFFERENTIAL DIAGNOSIS.

Tic douloureux has always to be considered when dealing with a complaint of pain in the face, though this diagnosis is often made when in actual fact the condition is not this malady at all. The essential features of tic douloureux are as follows. The pain is paroxysmal, the paroxysms lasting anything up to a minute and a half, and becoming more frequent in occurrence. There are complete remissions during which time there is no disturbance of sensation whatsoever, and the patient is normal in every respect. Each paroxysm is catastrophic in onset, the pain being variously described as darting, shooting, stabbing, tearing, burning, etc. The area affected corresponds anatomically to one or more divisions of the Vth cranial nerve, the second and third being the common ones. The pain is initiated by talking, eating, sneezing, shaving, or touching a certain spot which is constant in that particular individual and which has been termed by Patrick the 'trigger zone'. Paroxysms are frequently accompanied by vasomotor phenomena, such as flushing of the face, lachrymation, or slight watery

discharge from the nostril of the same side. The victim of major neuralgia adopts a typical attitude, and Dr. Adson regards this as of importance as a diagnostic feature. The voluble patient does not suffer from *tic douloureux*. A typical sufferer looks tired and worn and wears a mask-like expression. He sits in the chair with his head kept rigidly still and frequently wrapped in a shawl, and answers questions in the minimum number of words mumbled through the corner of the mouth, fearful lest the slightest movement bring on one of the dreaded attacks.

There is a type of pain which is deep-seated in the region of the root of the nose, upper maxilla, and malar bone, tending to spread back into the ear, mastoid, and down into the neck. Sluder described this syndrome, and considered it to be due to congestion or inflammation of the sphenopalatine ganglion secondary to infection of one of the paranasal sinuses. The pain is more or less continuous, and there is no impairment of sensation. Not much credence is given to this combination of symptoms as a definite clinical entity.

Hunt has described a type of facial pain affecting the external auditory meatus and radiating into surrounding parts which he attributes to inflammation of the geniculate ganglion of the VIIth cranial nerve. It is not paroxysmal. This again is not accepted by many authors.

Glossopharyngeal neuralgia, on the other hand, is quite a well defined clinical entity. The behaviour of the pain is very similar to that of *tic douloureux*, only the site being different. It is characterized by darting, shooting, burning, or scratching pain in the region of the angle of the jaw, tonsil, and pharynx. There is often a trigger zone which is situated in the supratonsillar fossa. The acts of talking, coughing, and especially swallowing may at any time bring on a paroxysm. The patient is thus rendered afraid to eat or even to drink. During my last month at the Mayo Clinic I was fortunate enough to see one of these cases. The patient had been so afraid of swallowing that for the month previous to her coming to the Clinic she had almost starved. The points of differentiation from *tic douloureux* are that with the mouth closed and at rest the whole face may be stroked without initiating an attack of pain, and that cocaineization of the supratonsillar fossa on the affected side abolishes the attacks so that the patient can eat and drink. The patient above mentioned was treated by cocaineization of the trigger zone for a week prior to operation in order to allow her to swallow and so to build up her depleted strength. Dr. Adson avulsed the glossopharyngeal nerve in the posterior fossa through a cerebellar approach. A fortnight after the operation she was completely free from pain.

Migraine is frequently accompanied by pain in the region of the eye, and there are often vasomotor disturbances too. However, the headache, and ocular disturbances such as 'steaming' and fortification spectra, make the diagnosis fairly straightforward.

Herpes, affecting most commonly the supra-orbital region, is accompanied by very severe pain. This is of a continuous type, and the eruption labels the case. Then there are a whole series of septic conditions which give rise to facial pain. This pain is throbbing and continuous, and does not conform to any definite anatomical area. Careful search will usually reveal some

septic focus, the eradication of which will produce a subsidence of pain, though it may be said that the wholesale drainage of sinuses and extraction of teeth without real evidence of definite pathology are only too frequently done. Such conditions as apical abscess, buried stumps, bony spurs, minute sequestra, granulomata, etc., may be at the bottom of the trouble, and must be excluded before a more serious course is elected. X rays may or may not show up any one of these lesions, but Melchior has pointed out that whenever one of these conditions is in an active state and is the true source of the referred pain a tender spot will be found opposite the alveolus. Infected sinuses will also be recognized by their appropriate signs and symptoms. With the modern use of block anæsthesia for dental extraction a temporary neuritis may develop. This is accompanied by severe pain, which soon subsides, but may be followed by anæsthesia in the area concerned.

Finally, there are cases in which there is actual pressure on some part of the trigeminal nerve. Probably the commonest of these is the acusticus tumour or neurofibroma of the auditory nerve. The usual complaint is increasing deafness, followed by vertigo, nausea, and vomiting, and on examination in a well established case there is usually corneal anæsthesia. However, on rare occasions facial pain is the first complaint, and the whole picture may very closely simulate major neuralgia. Such a case is reported by Parker.³⁴ Even in these cases, on close questioning it can usually be elicited that the patient had realized that his hearing on the one side had become defective some years previously, but so little did it bother him that he took no notice of it. Only with the onset of pain did he seek advice.

Intracranial aneurysms may be congenital or acquired, the congenital type being the more common (E. G. Fearnside³⁵ and Brain and Strauss³⁶). These occur most frequently on the circle of Willis or on its branches immediately after their origin, particularly on the anterior cerebral, anterior communicating, internal carotid, and middle cerebral arteries. An aneurysm in any of these four situations may or may not give rise to signs and symptoms of localizing value. Such signs and symptoms are headache, tinnitus, disturbance of vision due to pressure on the chiasm producing primary optic atrophy, hemianopsia, etc., ocular palsies due to pressure on the IIIrd, IVth, or Vth cranial nerves, and sometimes pain or anæsthesia in the first-division area of the Vth cranial nerve. Sometimes a bruit may be heard over the temporal region, though not so consistently as in the case of arteriovenous aneurysm between the internal carotid artery and the cavernous sinus, a condition resulting from trauma. Only too frequently the first indication of an intracranial aneurysm is its rupture.

Facial pain and paræsthesias may be a feature in cavernous sinus thrombosis. This condition results from some septic process of the face, nose, or sinuses, and is accompanied by œdema of the lids, chemosis of the conjunctiva, internal and external ophthalmoplegia, fever, rigors, etc.

Other lesions involving pressure on the Vth nerve, such as meningioma of the floor of the middle fossa, bony tumours, tumours of the pituitary, spread of malignant disease as from the pharynx (Woltman³⁷), have been instanced from the literature earlier on.

VIII. CONCLUSIONS.

In the nine cases which have been presented in detail, and in those quoted from the literature more briefly, certain facts common to all stand out. The pain is in the Vth-nerve area and rarely confined to one division only. It is fairly persistent in character, and is accompanied by alteration in sensation in a somewhat smaller part of the same area. This alteration of sensation is sometimes a subjective numbness, sometimes a tingling, burning, or feeling of stiffness; or, on the other hand, the change may only be discovered on examination, particularly a partial anæsthesia of the cornea. The motor root is usually affected too, as evidenced by paresis or paralysis of the muscles of mastication. Only when the growth spreads are other parts involved. Thus, the VIth nerve is one of the first to be affected; indeed, in some instances the external rectus muscle becomes weak before the masticatory muscles show any change, though always after the development of anæsthesia or paræsthesia. Then the IIIrd and IVth nerves show signs of pressure. These complications are due to a spread medialwards. If, on the contrary, the extension be backwards, posterior-fossa involvement becomes evident in the form of deafness, tinnitus, facial palsy, difficulty in swallowing, hemiatrophy of the tongue, shoulder weakness, or even signs of pressure on the cerebellum, sensory tracts, or pyramids, the latter giving contralateral signs or symptoms. But always, when the tumour is intrinsic, the order of events is pain, anæsthesia or paræsthesia, and weakness of the ipsilateral masticatory muscles. The order of onset of other signs and symptoms depends upon the direction of spread of the growth. It would appear that this applies to inflammatory lesions just as it does to neoplastic lesions. In the extrinsic tumours, such as *Case 2*, the youth with the overflowing pituitary tumour, and Benson's case, symptoms and signs due to pressure on structures other than the ganglion may develop first. Such cases have been discussed when the differential diagnosis was considered. A meningioma of the middle fossa may arise from immediately above the ganglion, resulting in a syndrome resembling that of intrinsic tumours. Such were the cases of Giani, Sachs, and De Martel, and *Case 5* of this series.

So in conclusion it may be said that there is a set of signs and symptoms which can reasonably be interpreted as indicating a progressive tumiform lesion either within the Gasserian ganglion itself or in its immediate vicinity. Differentiation between neoplastic and inflammatory processes cannot at the moment be made, though the law of averages is greatly in favour of the former. To recapitulate, the picture is as follows:—

The very first symptom is pain in the Vth-nerve area affecting usually more than one division. It is more or less continuous in character, and progressive in severity and persistence. Arising either simultaneously with the original onset of pain or very shortly afterwards is a subjective alteration in sensation in the same area. This may be in the form of numbness or paræsthesia. Occurring at about the same time in a large proportion of the cases is a paresis of the masticatory muscles. Following this, a series of signs and symptoms may develop depending on the direction of spread, and by the time the patient presents himself for advice he may show involvement

of the ocular nerves, facial nerve, auditory nerve, or even of the IXth, Xth, XIth, or XIIth nerves. However, a careful search for the sequence of events from the very first complaint will lead one to conclude that the lesion began in the ganglion or in the middle fossa in its immediate vicinity.

The advisability of operating when posterior-fossa symptoms are present is a matter of question. It is certain that, benign though the growth may be, it cannot be removed in its entirety from either the posterior-fossa or middle-fossa approach. Nevertheless, if the pain is very severe, as was the case in all the present series, something must be done. Section of the posterior root with removal of as much tumour as possible offers the best chance of relief.

SUMMARY.

1. Seven cases of tumour and two of inflammatory lesions of the Gasserian ganglion are presented.

2. The development and anatomy of the ganglion are described.

3. The literature on tumours and inflammations of the ganglion is reviewed.

4. The complete history, treatment, and progress of each of the nine cases are given.

5. In considering the pathology of the tumours it is opined that four out of the seven cases are true intrinsic tumours of the ganglion. It is also suggested that some of the tumours described in the literature as 'endothelioma' of the ganglion are true intrinsic tumours, for it is pointed out that just as meningiomata, which are still often described as "endothelioma arising from the lining cells of the dura", do not infiltrate the brain, so there is no reason to assume that they invade the ganglion, which is surrounded by arachnoid and dura in the same way as the brain is surrounded.

6. The present-day operation, with its variations, is described.

7. The many other conditions producing pain in the Vth-nerve area are considered, and the differential diagnosis is discussed.

8. A constant symptom-complex is developed from which it is considered possible to diagnose these lesions. The all-important point is the sequence of events.

It was my good fortune through the tenure of a Rockefeller Travelling Fellowship to spend the year 1929 visiting various hospitals and clinics in North America and Canada. The first eight months of this time were passed at the Mayo Clinic working in the Department of Neurosurgery of which Dr. A. W. Adson is the head. It is to him that I am indebted for the privilege of presenting the nine cases which form the basis of this work, and I am particularly grateful to him for his ever-ready advice and teaching. I am indebted to Dr. Robertson, Chief of the Department of Pathological Anatomy, for permission to use the laboratories and museums, and to Dr. Kernohan, Neuropathologist at the Clinic, for kindly advice. Finally, I would express my gratitude to the Mayo Clinic for affording me access to the histories and records.

REFERENCES.

- ¹ SHILDEN, "Gasserian Ganglion Tumors", *Jour. Amer. Med. Assoc.*, 1921, lxxvii, 700.
- ² BARTELMIEZ and EVANS, quoted by C. H. Frazier and E. Whitehead.
- ³ FRAZIER, C. H., and WHITEHEAD, E., "Morphology of the Gasserian Ganglion", *Brain*, 1925, xlviii, 458.
- ⁴ LOCKHART, R. D., "The Aural Relations of the Gasserian Ganglion with Reference to a New Method of Surgical Approach", *Jour. of Anat.*, 1927, lxii, 105.
- ⁵ FRAZER, J. ERNEST, *Anatomy of the Human Skeleton*. London: J. & A. Churchill.
- ⁶ Quoted by Gjertz and Helleström.
- ⁷ FISCHER, *Charité-Annalen*, 1863, x, part 2.
- ⁸ KROGIUS, "Du Traitement chirurgical des Tumeurs de la Fosse latérale moyenne du Crâne", *Rev. de Chir.*, 1896, xvi, 434.
- ⁹ DERCUM, KEEN, and SPILLER, "Endothelioma of the Gasserian Ganglion", *Jour. Amer. Med. Assoc.*, 1900, xxxiv, 1025.
- ¹⁰ Quoted by Dereum, Keen, and Spiller.
- ¹¹ GIANI, "Über einen Fall von Endotheliom des Gang. Gass.: anatomische, pathologische, und klinische Beobachtung", *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, xix, 452.
- ¹² SACHS, "Tumors of the Gasserian Ganglion", *Ann. of Surg.*, 1917, lxvi, 152.
- ¹³ FRAZIER, "An Operable Tumor involving the Gasserian Ganglion", *Amer. Jour. Med. Sci.*, 1918, clvi, 483.
- ¹⁴ GJERTZ and HELLERSTRÖM, "Tumeur du Ganglion de Gasser", *Acta Med. Scand.*, 1925-6, lxiii, 7.
- ¹⁵ ALTMANN, "Primary Tumours of the Gasserian Ganglion", *Beitr. z. pathol. Anat. u. allgem. Path.*, 1928, lxxx, 361.
- ¹⁶ RUSSELL, ETHEL C., "Two Primary Tumors of the Gasserian Ganglion", *Jour. Amer. Med. Assoc.*, 1925, lxxxiv, 413.
- ¹⁷ RAND, C. W., "Tumor of Left Gasserian Ganglion", *Surg. Gynecol. and Obst.*, 1925, xl, 49.
- ¹⁸ DE MARTEL, *La Thérapentique des Tumeurs cérébrales. Technique chirurgicale. Rapport présenté à la Société internationale de Chirurgie, Rome, 1926, April.*
- ¹⁹ BENSON, R. L., "An Unusual Tumour involving the Hypophysis and Gasserian Ganglion", *U. S. Fed. Bureau Med. Bull.*, 1927, iii, 236.
- ²⁰ NONNÉ, quoted by Henry Veits.
- ²¹ MOTT, quoted by Henry Veits.
- ²² VEITS, H., "Syphilis as an Etiological Factor in Trigeminal Neuralgia", *Albany Med. Ann.*, 1920, xli, 345.
- ²³ SMITH, CARROL, "Abscess of Gasserian Ganglion complicating Mastoiditis", *Ann. of Otol. Rhinol. and Laryngol.*, 1925, xxxiv, 938.
- ²⁴ GRADENIGO, *Arch. f. Ohrenheilk.*, 1904, lxii, 256.
- ²⁵, ²⁶ VOGAL and KUICK, quoted by Friedenwald.
- ²⁷ WEINER, H. S., "Tic Douloureux Cured by Mastoidectomy", *Arch. of Otolaryngol.*, 1927, v, 344.
- ²⁸ WHEELER, J. M., "Paralysis of the Sixth Cranial Nerve in Otitis Media", *Jour. Amer. Med. Assoc.*, 1918, lxxi, 1718.
- ²⁹ PERKINS, "Abducens Paralysis in Purulent Otitis Media", *Ann. of Otol. Rhinol. and Laryngol.*, 1910, xix, 692.
- ³⁰ WEINER, quoted by Giani.
- ³¹ FRIEDENWALD, "Unusual Forms of Extension in Purulent Otitis Media with Special Reference to Involvement of Cranial Nerves", *Laryngoscope*, 1923, xxxiii, 820.
- ³² DENCH, E. B., "Acute Mastoiditis with Unusual Symptoms", *Ann. of Otol. Rhinol. and Laryngol.*, 1916, xxv, 672.
- ³³ HEAD, HENRY, "Pathology of Herpes Zoster", *Brain*, 1900, xxiii, 501.
- ³⁴ PARKER, H. L., "Tumors of Nervus Acusticus", *Arch. of Neurol. and Psychiat.*, xx, 309.
- ³⁵ FLAARNSIDES, E. G., *Brain*, 1916, xxxix, 224.
- ³⁶ BRAIN and STRAUSS, *Recent Advances in Neurology*, 1929.
- ³⁷ WOLTMAN, H. W., *Malignant Tumours of the Naso-pharynx with Involvement of the Nervous System*.

INTESTINAL OBSTRUCTION COMPLICATING POSTERIOR GASTROJEJUNOSTOMY:

A CASE OF INTERNAL STRANGULATION OF THE SMALL INTESTINE
BY THE AFFERENT LIMB.

By GEORGE ARMITAGE,

SENIOR SURGICAL TUTOR IN THE UNIVERSITY OF LEEDS.

ILEUS, or intestinal obstruction, is one of the unfortunate sequelæ which the operation of posterior gastrojejunostomy inherits as a permanent legacy. It is rare, occurring at any time after operation, being immediate, early, or late in development. There may be either a physiological or a pathological explanation of the obstruction. The significance of the former should be remembered especially when performing the operation, the importance of which will be seen later, and also if called upon to treat a case of immediate or early post-operative ileus occurring within a period of three weeks of the operation. After that time it is true to say that a physiological explanation of the obstruction can be definitely ruled out, whereas before that interval of time has elapsed it may be either physiological or pathological.

PHYSIOLOGICAL CONSIDERATIONS.

Interference with the normal physiological movements of the jejunum may occur following what is generally regarded, in the absence of definite contra-indication, as the ideal method of uniting the stomach and jejunum—

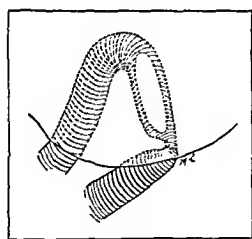


FIG. 91.—Diagram showing kink immediately distal to the stoma in a posterior vertical no-loop (isoperistaltic) gastrojejunostomy. Note the interruption of the circular muscle-fibres. (After Cannon, modified.)

namely, the posterior vertical no-loop gastrojejunostomy. The stoma in the stomach is placed as nearly vertical as possible in the line of the right margin of the cardiac orifice, its lower end reaching the greater curvature. In the jejunum the stoma is situated just on what will be the gastric side of the antimesenteric border, thereby causing the jejunum to apply itself more closely to the stomach, immediately distal to the duodenojejunal flexure. As a result the circular muscle-fibres of the jejunum are cut across for a distance corresponding to the length of the stoma—that is, about two to three inches. This interruption of their continuity destroys the local reflex of the bowel wall described by

Bayliss and Starling,¹ whereby stimulation of the bowel at any point causes contraction behind and relaxation in front of the point stimulated. Cannon² pointed out that as a result there is not present in the bowel wall the

necessary mechanism to straighten out a kink which may occur immediately distal to the stoma. Contraction of the circular muscle-fibres then results only in shortening of the bowel without accompanying distal dilatation. Almost always the kink occurs immediately distal to the stoma in the efferent loop (*Fig. 91*).

When the normal freedom and ease of movement of the various coils of the small intestine is understood, it will be readily appreciated how the anchoring of one loop may produce a kink. Evidence of this is commonly seen in the cases of acute and chronic obstruction resulting from the presence of pathological bands and adhesions. In addition, the normal movements of the stomach may actually produce kinking of the jejunum. If, at operation, there appears to be any tendency towards kinking, particularly of the efferent loop, this may be stitched to the stomach beyond the limits of the stoma—a practice Hartmann³ advocated with both the afferent and efferent loops in anterior gastrojejunostomy, and one which has a sound physiological basis in any gastrojejunostomy.⁴

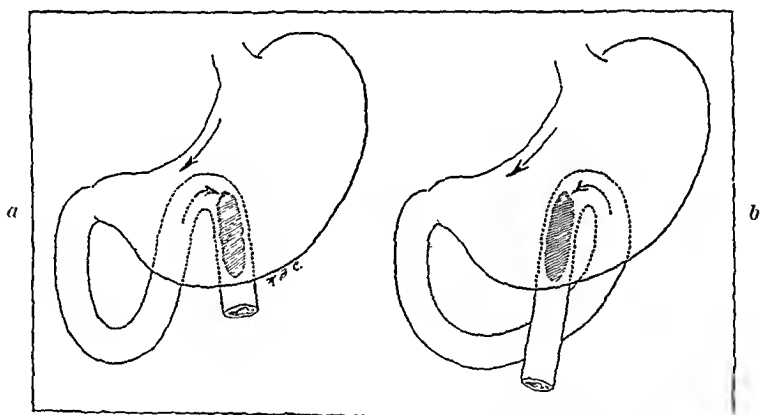


FIG. 92.—*a*. The antiperistaltic posterior method of performing gastrojejunostomy :
b. The isoperistaltic method of performing posterior gastrojejunostomy.

Whether the antiperistaltic method (*Fig. 92 a*) advocated by Bier⁵ and the Mayos,⁶ or the isoperistaltic method (*Fig. 92 b*) advocated by Moynihan,⁷ is performed does not appear to affect the incidence of either physiological or pathological ilcus as judged by experience and results.

It is opportune to mention here the so-called 'vicious circle' associated with posterior gastrojejunostomy. As the operation is now performed without the formation of a loop on the afferent side of the stoma, the occurrence of vomiting due to the development of a 'vicious circle' is enormously reduced. In some cases the cause may be mechanical obstruction due to faulty technique, but in the majority of cases no mechanical explanation is found. Binnie⁸ stated that 'vicious-circle' vomiting depended more upon the loss of contractile power of the stomach wall than upon any other factor. This statement is as significant to-day as it was nearly forty years ago. In any extensive series of gastrojejunostomies there will, not infrequently, be cases

which have a troublesome and stormy post-operative period commencing with vomiting any time within ten days of the operation. The great majority, if not all, of these cases either recover spontaneously or respond to such remedial measures as absolute starvation per os, fluids being administered rectally, combined with lavage of the stomach, and in the most severe cases the assumption of the prone position advocated by Schnitzler¹⁰ in 1895, the importance and practice of which was re-stressed by Borchgrevink¹¹ in 1913.

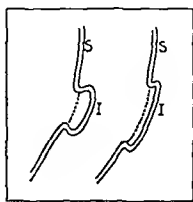


FIG. 93.—Diagram showing the effect of dilatation of the stomach upon the stoma of a gastrojejunostomy. (After Cannon.)

It is only by early recognition and treatment of this condition that the more serious complication of acute dilatation of the stomach may be prevented, a condition which, in my opinion, differs only in degree from that of 'vicious-circle' vomiting due to loss of contractile power of the stomach wall. Cannon¹² clearly pointed out with the aid of a simple diagram (Fig. 93) that the effect of distension of the stomach was to flatten the jejunum against it, thereby obstructing or even completely obliterating the stoma between them.

The old theories advanced to explain the occurrence of 'vicious-circle' vomiting—regurgitation of bile into the stomach, obstruction of the third part of the duodenum by the superior mesenteric vessels, and others—can be dismissed.

The physiological causes may be briefly summarized as follows:—

1. Interference with the normal movements of the jejunum as a result of: (a) Division of the circular muscle-fibres over a length of bowel corresponding to that of the stoma, thereby destroying the normal reflex described by Bayliss and Starling; (b) Mere mechanical fixation of a loop of jejunum; (c) The effect of the stomach movements upon those of the jejunum.

2. Distension of the stomach due to loss of contractile power of the stomach wall.

PATHOLOGICAL CONSIDERATIONS.

Temporary paralytic ileus may occur as a result of trauma or low-grade infection due to faulty technique—for example, rough handling, too vigorous application of clamps, or soiling of the operation field due to the escape of stomach or jejunal contents.

Gross mechanical ileus occurs in the following ways:—

1. Adhesions involving either the afferent or the efferent limb causing interference with motility or definite kinking. Cicatricial contracture of the transverse mesocolon as a result of operative trauma may produce ileus.¹³

2. Herniation of the small intestine into the lesser sac through the opening in the transverse mesocolon. This is an uncommon event nowadays, because the importance of stitching the margins of the opening in the mesocolon to the stomach (Sherren¹⁴), to the jejunum (Moynihan¹⁵), or to the anastomotic line picking up both stomach and jejunum (Bier¹⁶), has been stressed, and the closure of this opening is now universally practised. Examples of this type of case are recorded by Moynihan,¹⁷ Ashhurst,¹⁸ and

Hartmann.¹⁹ It is interesting, in view of the author's case described below, that in 1906 Moynihan²⁰ wrote: "After the posterior no-loop operation with suture of the mesocolon to the jejunum it [internal strangulation] has never occurred."

3. Retrograde intussusception of the jejunum towards or actually through the stoma into the stomach. A complete summary of this condition was given in this JOURNAL by Hamilton Drummond²¹ in which he pointed out that it might occur at any time and after any variety of gastrojejunostomy ;

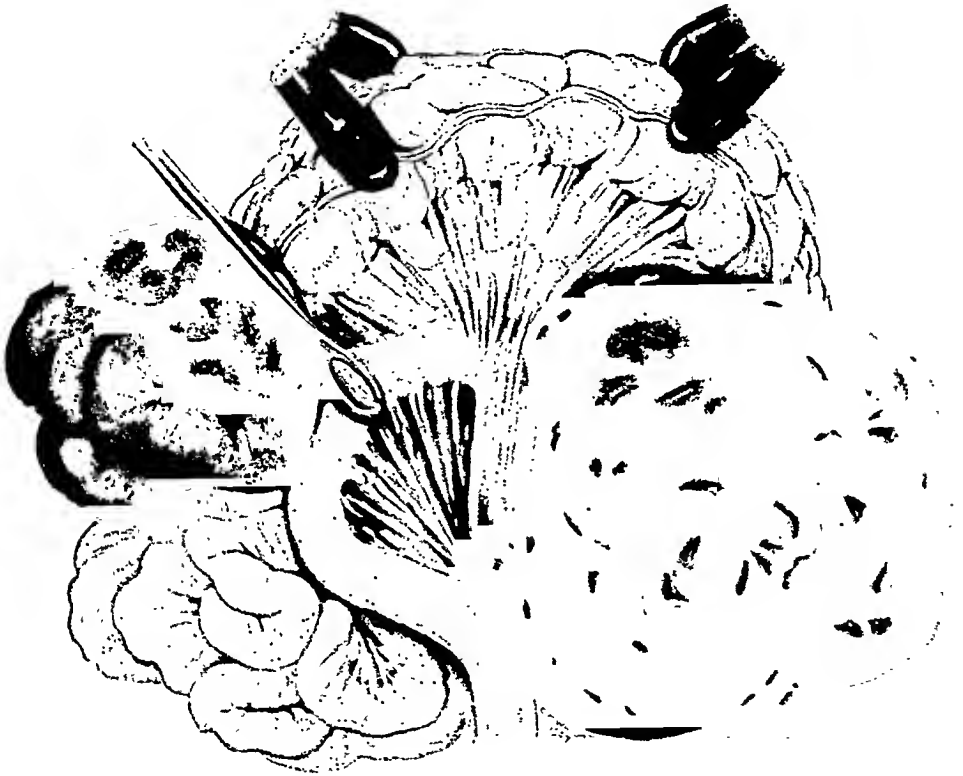


FIG. 94.—Showing the short afferent limb of the gastrojejunostomy above which the small intestine is passing between it and the under aspect of the transverse mesocolon. On the left, above is seen the dilated small intestine proximal to the obstruction, and below the collapsed small intestine distal to the obstruction. On the right is seen the distended gangrenous small intestine. (Drawn at autopsy after replacing the intestine in the position found at operation.)

that it was a well recognized condition ; that the diagnosis should be made ; and that there was no known method of preventing its occurrence. The situation remains unchanged. It is interesting to note that I have been unable to find any references to cases of this kind since the publication of Drummond's article over six years ago.

4. The establishment of the 'vicious circle' as a result of pathological lesions has already been noted.

5. Internal strangulation of the small intestine by the short efferent limb of jejunum, i.e., that segment of the bowel between the duodenojejunal flexure and the commencement of the gastrojejunostomy stoma.

CASE REPORT.

W. D., age 45 years, an iron moulder, was admitted to hospital in a moribund condition on Dec. 8, 1927.

HISTORY.—The history was that the patient commenced with sudden acute abdominal pain four days previously. The pain was of a colicky nature, coming on spasmodically and causing him to double up. There had been persistent vomiting from the first, the patient having been unable to keep "anything on his stomach". At first the vomit was gastric, later bilious, and finally foul-smelling and definitely faecal. Constipation had been present, but it was not possible to elicit positive evidence of absolute constipation, although this was not doubted.

ON EXAMINATION.—Marked and general distension of the abdomen was present. Percussion revealed a tympanitic note in front with dullness in the flanks. Over the right rectus was seen the scar of a previous operation wound which, we elicited from the records, had been performed by the late Mr. Walter Thompson for chronic anterior duodenal ulcer in 1914—i.e., thirteen years previously. The operation consisted of removing the appendix, enfolding the ulcer, and performing a posterior vertical no-loop gastrojejunostomy, since when the patient had enjoyed good health up to the present acute illness. A diagnosis of acute intestinal obstruction of a high type was made, and laparotomy decided upon under gas- and -oxygen anaesthesia. Preliminary washing out of the stomach was carried out.

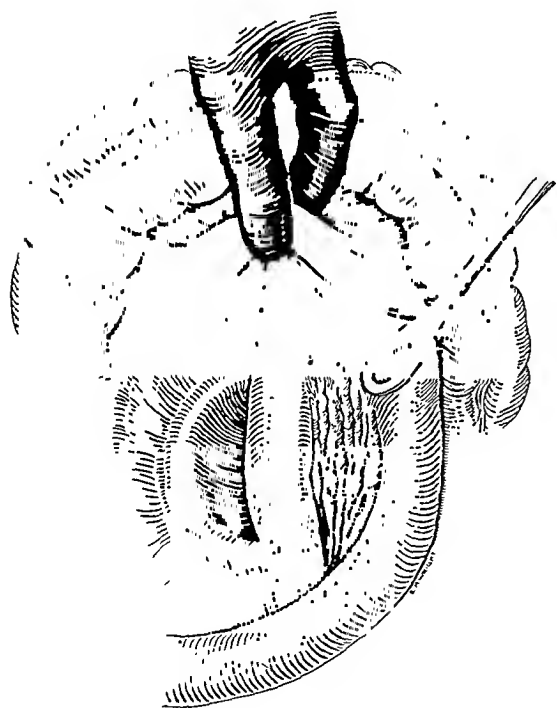


FIG. 95.—To show the boundaries of the opening through which the small intestine had herniated after removing the small intestine depicted in Fig. 94.

the previous incision. Serosanguinous fluid escaped from the peritoneal cavity, and the condition depicted in Fig. 94 was observed upon lifting up the great omentum and the transverse colon.

At least ten feet of the small bowel had passed from right to left through an aperture bounded above by the under aspect of the mesocolon, in front by the gastrojejunal anastomosis, behind by the posterior abdominal wall covered by peritoneum, and below by the short afferent limb running from the duodenojejunal flexure to the stoma. The boundaries are well illustrated in the accompanying drawing (Fig. 95).

OPERATION.—The abdomen was opened through the scar of

The length of the afferent limb which was acting as a tight constricting agent was $2\frac{1}{4}$ in. Without any serious difficulty it was possible, probably on account of the non-viable state of the gut, to reduce the whole of the herniated bowel from left to right through the opening described above. No improvement whatever took place in its condition after reduction, but on account of the patient's desperate condition further treatment was impossible. He succumbed six hours later.

POST-MORTEM.—At autopsy the condition was verified, reproduced, and the illustrations were drawn. The old duodenal ulcer was healed, and the gastrojejunostomy admitted the tips of three fingers.

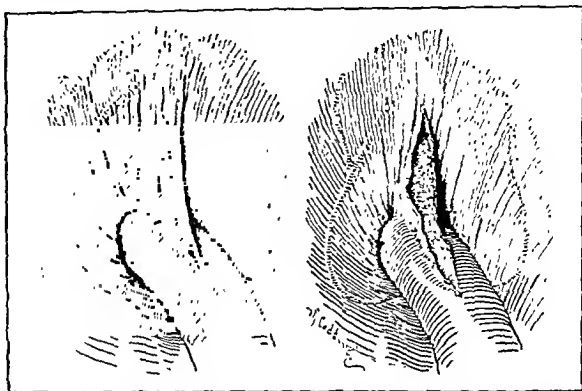


FIG. 96.—Showing a well-marked suspensory ligament of Treitz with attachment to the jejunum of at least $1\frac{1}{2}$ to 2 in. distal to the duodenojejunal flexure. Resection of above in order to ensure that the jejunal stoma be placed as near the duodenojejunal flexure as possible.

Similar cases have been recorded by Gray,²² Barker,²³ and Gordon.²⁴ A comparable case occurring in connection with anterior gastrojejunostomy and associated with a twist at the anastomotic opening has also been reported by W. J. Mayo.²⁵

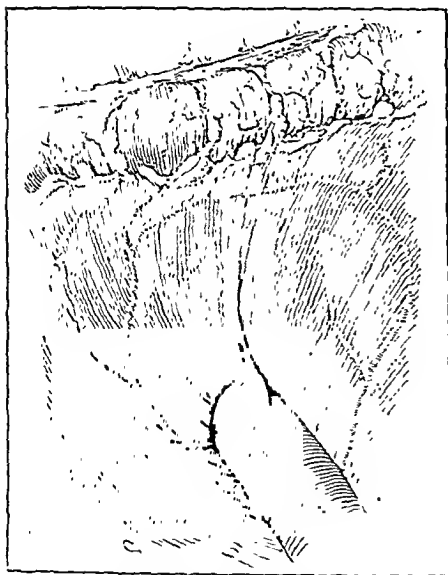


FIG. 97.—Showing a suspensory ligament of Treitz of average development.

COMMENT.

This complication of a posterior gastrojejunostomy is an exceedingly rare one. I have only been able to find the reports of the three cases mentioned above. That it may occur has long been recognized, the herniated bowel passing either from right to left or from left to right through the stoma. It is a potential happening in all cases; the chances of its occurrence being reduced enormously by ensuring that the afferent limb is as short as possible, measuring less than 1 to $1\frac{1}{2}$ in. It can be absolutely prevented by stitching the afferent limb to the mesocolon, a procedure which is difficult to carry out. If this is done, great care must

be exercised to prevent kinking, and to ensure that important vessels in the mesocolon are not pricked. This procedure has not been carried out as a routine in the performance of a posterior gastrojejunostomy, surgeons relying

entirely upon the short length of the afferent limb. How can this be obtained in all cases? First, by resecting the suspensory ligament of Treitz in cases where it is well developed and encroaches upon the jejunum distal to the flexure (*Fig. 96*). In *Fig. 97* is shown a suspensory ligament of Treitz of average development.

W. J. Mayo²⁶ stressed the importance of this, and it is confirmed by Bier.²⁷ It is then possible in all cases to ensure that the stoma be situated immediately distal to the flexure, thereby reducing the length of the afferent limb to a minimum. Many surgeons do not use clamps, particularly on the jejunum, which it is well to remember were first used in this operation by Littlewood,²⁸ the details of which formed the subject of a paper read before the Leeds and West Riding Medico-Chirurgical Society on Oct. 19, 1900. Littlewood's original work was the subject of a letter written by his colleague, Ward,²⁹ to the *Lancet* in 1906. If a clamp is not used on the jejunum, it is technically easier to place the stoma nearer to the duodenojejunal flexure. If a clamp is used, this result will be more readily obtained by applying the clamp with the tips of the blades towards the flexure.

SUMMARY.

1. The importance of the interference with the normal physiological movements of the jejunum in any gastrojejunostomy should always be carefully borne in mind.

2. Mechanical ileus complicating a posterior gastrojejunostomy due to the variety of causes mentioned may happen at any time during the patient's life.

3. A rare and fatal case of internal strangulation of the small intestine by the short afferent limb is recorded. It is a potential happening in all cases unless the short limb is stitched to the mesocolon.

4. The rarity of occurrence, especially noted in recent years, must be due to the extremely short length of the afferent limb, and not to the closure of the opening described above—a procedure which is not practised as a routine.

I am indebted to Professor J. F. Dobson, under whose care this patient was admitted, for kind permission to publish the case. My thanks are due to Miss Wright and Miss Codding for the illustrations.

REFERENCES.

- ¹ BAYLISS and STARLING, *Jour. of Physiol.*, 1899, xxiv, 110.
- ² CANNON and BLAKE, *Ann. of Surg.*, 1905, xli, 701.
- ³ BICKHAM, *Operative Surgery*, iv, 322.
- ⁴ CANNON, *Boston Med. and Surg. Jour.*, 1909, cxi, 720.
- ⁵ BIER, BRAUN, KUMMEL, *Chirurgische Operationslehre*, iii, 116.
- ⁶ MAYO, W. J., *Ann. of Surg.*, 1906, xliii, 537.
- ⁷ MOYNIHAN, *Abdominal Operations*, i, 188.
- ⁸ BINNIE, *Treatise on Regional Surgery*, 1893, ii, 126.
- ⁹ BINNIE, *Brit. Med. Jour.*, 1893, i, 1149.
- ¹⁰ SCHNITZLER, *Wein. klin. Rundsch.*, 1895, No. 35.

- ¹¹ BORCHGREVINK, *Surg. Gynecol. and Obst.*, 1913, June, 662.
- ¹² CANNON, *Ann. of Surg.*, 1905, 699.
- ¹³ SOUTHAM, *Lancet*, 1928, i, 41.
- ¹⁴ CHOYCE, *System of Surgery*, ii, 423.
- ¹⁵ MOYNIHAN, *Abdominal Operations*, i, 184.
- ¹⁶ BIER, BRAUN, KUMMEL, *Chirurgische Operationslehre*, iii, 115.
- ¹⁷ MOYNIHAN, *Lancet*, 1906, i, 1345.
- ¹⁸ CHOYCE, *System of Surgery*, ii, 432.
- ¹⁹ *Ibid.*
- ²⁰ MOYNIHAN, *Lancet*, 1906, i, 1345.
- ²¹ DRUMMOND, HAMILTON, *Brit. Jour. Surg.*, 1923, xi, 79.
- ²² GRAY, *Lancet*, 1904, ii, 526.
- ²³ MOYNIHAN, *Abdominal Operations*, i, 209.
- ²⁴ GORDON, *Lancet*, 1905, ii, 1477.
- ²⁵ MAYO, W. J., *Ann. of Surg.*, 1902, xxxvi, 241.
- ²⁶ MAYO, W. J., *Ibid.*, 1908, xlvii, 1.
- ²⁷ BIER, BRAUN, KUMMEL, *Chirurgische Operationslehre*, iii, 108, 109.
- ²⁸ LITTLEWOOD, *Lancet*, 1901, i, 1817.
- ²⁹ WARD, *Ibid.*, 1906, i, 1347.

*SHORT NOTES OF
RARE OR OBSCURE CASES*

**A CASE OF UNILATERAL POLYCYSTIC KIDNEY IN A CHILD,
AGE ONE YEAR AND EIGHT MONTHS.**

BY CECIL P. G. WAKELEY,
SURGEON TO KING'S COLLEGE HOSPITAL, LONDON.

CONGENITAL polycystic disease of the kidneys in the adult is, of course, a familiar condition, but when occurring unilaterally in an infant must be regarded as a pathological curiosity. Out of a total of 1411 cases examined in the post-mortem register of the Royal Hospital for Sick Children, Glasgow, between the years 1915-25, Young¹ could find one case only, and that a bilateral one. The post-mortem records of the Belgrave Hospital for Children from 1909-29 reveal 2 cases of polycystic kidney out of a total of 2110 cases. Kidd² collected 149 recorded cases of the condition, and found that 9 only were unilateral, and of these 6 occurred on the left side and 3 on the right. Willan,³ in an analysis of 22 cases which came under his personal cognizance, did not find one under the age of 10 years.

The division of the disease into three stages, the latent, the tumour, and the uræmic, is purely arbitrary, since a latent period may be absent.

The following is the report of a recent case:—

HISTORY.—Henry J. C., age 20 months, was admitted under Dr. Still at King's College Hospital on April 6, 1929, with a large abdominal tumour which was first noticed by the child's mother a few months after birth. He was the third in the family, there being two older children of 12 and 14 respectively. Labour was normal and the child weighed 10 lb. at birth. He was breast-fed for over a year.

ON EXAMINATION.—On examining the abdomen a large smooth tumour was found to occupy the whole of the right side, extending upwards to the liver margin, just across the mid-line, and below into the iliac fossa. The tumour was of firm consistency and felt like rubber.

Urine: Specific gravity 1020; no casts; about 4 pus cells per field and 2 blood-corpuscles per field. Blood-urica, 39 mgrm. per 100 c.c.

As the tumour was growing fairly rapidly and was causing definite obstruction to the colon, it was decided to operate.

OPERATION.—Nephrectomy was performed under open ether on April 22, 1929. A right paramedian incision was made through the skin and subcutaneous tissues extending from the costal margin to just above the symphysis pubis. The inner border of the rectus sheath was opened and the rectus muscle displaced outward, the posterior sheath being carefully incised.

The peritoneum was stripped off a large renal tumour by gauze dissection, which proved to be quite easily performed. A very large solid renal tumour was exposed and delivered into the wound. There were many aberrant vessels, and the pelvis of the kidney was dilated. The vessels were divided between ligatures, and the ureter was dissected down to the brim of the pelvis and then ligatured. The kidney was then removed. The peritoneal cavity was explored, the left kidney palpated and found to be normal. The liver, spleen, and pancreas were examined and no evidence of further cystic disease was discovered. There was practically no bleeding, and the pulse and blood-



FIG. 98.—Posterior surface of the kidney, showing dilated pelvis. ($\times \frac{3}{2}$)

pressure remained constant throughout the operation. The posterior sheath of the rectus was sutured, the rectus muscle was allowed to fall back into place, and its anterior sheath was then sutured. The skin was approximated with interrupted silkworm-gut sutures. A small drainage tube was inserted for forty-eight hours. The wound healed by first intention, and the stitches were removed on the eighth day after the operation.

SUBSEQUENT HISTORY.—The operation did not seem to upset the child in the least, as he continued to gain weight each week while in hospital. An examination on May 5, 1929, proved the urine to be normal.

May 10, 1929: blood-urea, 30 mgrm. per 100 c.c. May 15: blood-cholesterol, 160 mgrm. per 100 c.c. May 22: blood-urea, 21 mgrm. per 100 c.c. May 23: blood-count—red corpuscles, 4,560,000 per c.mm., 91 per cent of normal; hæmoglobin, 70 per cent of normal; colour index, 0.77 per cent; leucocytes, 29.0 per cent; large hyaline mononuclears, 3.5 per cent. May 24: an examination of the urine showed that the slight deposit contained a few leucocytes and a few epithelial cells. The culture was sterile.

The child was discharged on May 26 in excellent health, and has been kept under constant observation, his last visit to the hospital being in January, 1930. There was no evidence of enlargement of the left kidney. The

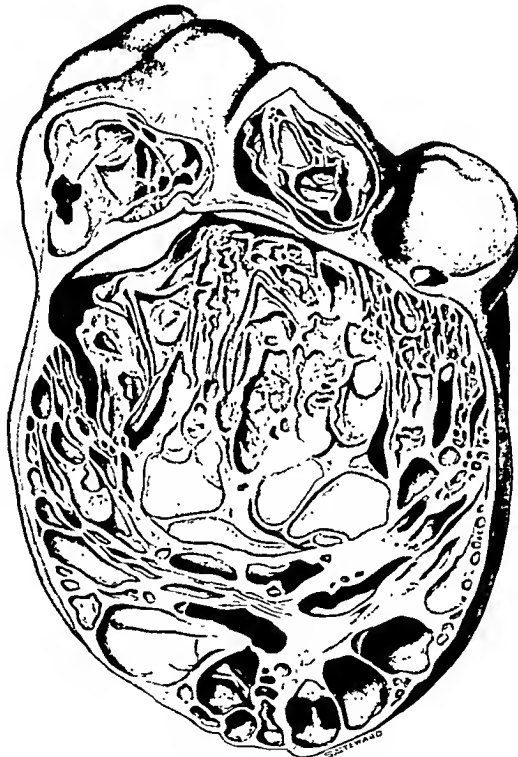


FIG. 99.—Vertical section through the kidney, showing the numerous cystic spaces. ($\times \frac{1}{2}$.)

examination of the urine was as follows: specific gravity, 1012; no deposit; culture sterile; blood-urea, 20 mgrm. per 100 c.c.

This case appears to be one of unilateral congenital cystic disease of the kidney, but it may be said that the other kidney, although showing no sign of disease at present, may do so at a later date.

PATHOLOGICAL REPORT.—The tumour measured 7 in. by 5 in. and weighed 50 oz. (*Fig. 98*). It is seen to be reniform in shape with definite cystic dilatations on its surface. The pelvis of the kidney was definitely dilated. On section (*Fig. 99*) no normal renal tissue whatever could be seen. The kidney was occupied by numerous cystic spaces varying considerably

in size. Some of these spaces contained fluid; others contained a colloid substance.

Microscopical Report.—The microscopical report was as follows:—

“There was no normal kidney tissue present, the normal structure being completely replaced by a number of cysts of size varying from 1 mm. to 5 mm. in diameter lying in a fibrous stroma. The type of epithelium lining the cysts showed considerable variation (*Fig. 100*). In some of the cysts the epithelium was definitely columnar, the cells having large vesicular nuclei situated towards the base of the cells. In other cysts, including the smallest, the

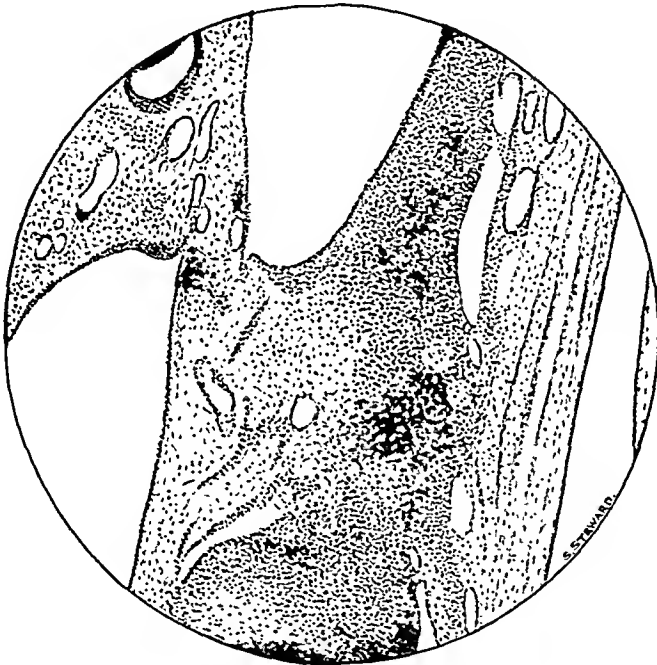


FIG. 100.—Microscopical section of the tumour. ($\times 45$.)

epithelium was very flattened, and the cells resembled endothelial cells. All intermediate types of cell were present. In all the cysts the epithelium was only a single cell thick. The stroma was a loose fibrous stroma showing areas of hyaline degeneration. Hæmorrhage, mostly recent, had occurred. Some pigment, chiefly within phagocytes, was present. The stroma showed areas of infiltration with inflammatory cells, including large numbers of plasma cells.”

REFERENCES.

- ¹ YOUNG, R. F., *Brit. Jour. Surg.*, 1924-5, xii, 244.
- ² KIDD, F., *Urinary Surgery*, 212.
- ³ WILLAN, R. J., *Newcastle Med. Jour.*, 1928, viii, 117.

CASE OF NON-ROTATION OF THE INTESTINE WITH CONGENITAL ABSENCE OF RADIUS.

By J. W. GEARY GRANT,

SURGEON TO THE ROYAL INFIRMARY, CARDIFF.

FAILURE of the second stage of rotation of the mid-gut loop is a comparatively rare condition; Dott¹ in 1923 had collected forty-eight cases from the literature, including three personal observations. Two of these were in infants, and probably many cases of this anomaly occur which never reach adult life. Failure of the third stage—descent of the cæcum to the right iliac fossa and fixation—is by no means rare, varying from minor degrees of mobility of the cæcum and descending colon to a cæcum and ascending colon provided with a complete mesentery. The symptoms to which these cases give rise, the conditions they mimic, and the appropriate treatment, have formed the subject of a very full and important paper by Waugh.²

The following case, exhibiting complete failure of the second stage of rotation, and accompanied by another rather rare congenital malformation, appears worth recording.

T. H. H., miner, age 32, a patient of Dr. W. Watkins, of Tonypandy, was diagnosed as suffering from appendicular colic.

HISTORY.—He was the eldest of a family of ten, and appears to have had no trouble until July, 1929, when he began to have attacks of generalized pain in the lower half of the abdomen, coming on a half to one hour after meals, lasting for about one hour, and described as agonizing. The bowels acted regularly without aperients; the



FIG. 101.—Skiagram showing congenital abnormality of arm and hand.

tongue was clean, and the pulse and temperature were normal.

There is also congenital absence of the left radius with the exception of the head and neck, absence of the left thumb, first metacarpal, and corresponding carpal bones (*Fig. 101*).

OPERATION (Sept. 5, 1929).—A muscle-splitting incision was made to remove the appendix, under general anaesthesia. A search with the finger failed to find the cæcum, and the abdomen was then opened by a right paramedian incision with its centre opposite the umbilicus. The whole of the small intestine was found to occupy the right side of the abdomen. After a brief search the right colon was found—comprising the cæcum, ascending

and transverse colon—on the left side, passing obliquely upwards to the fixed splenic flexure. The splenic flexure and descending colon occupied of course their normal positions, but the sigmoid, which formed a short loop, was attached to the right colon by omental bands and fine membranous adhesions. The ileum entered the cæcum on the right side. The duodenum formed a U-shaped loop around the head of the pancreas, then passed directly into the jejunum, uncrossed by and lying to the right of the colon. (Fig. 102.)

After removing the appendix, which was $2\frac{1}{2}$ in. long and normal in appearance, and dividing the bands uniting the right colon to the sigmoid, the midgut was rotated through 180° and a portion of colon corresponding to the hepatic flexure was brought across the root of the mesentery of the small intestine and fixed with a catgut suture to the posterior parietal peritoneum over the right kidney, two more stitches fixing the cæcum and ascending colon in their normal positions, as in Waugh's operation. The small intestine was pushed over to the left, care being taken that the proximal loops of jejunum should run to the left as normally, the terminal ileum entering the cæcum now on the left side; a restoration of the parts approximately to the normal appeared to have been obtained. Owing to the very short attachment of the mesentery—for in these cases the duodeno-colic isthmus formed by the approximation of the duodenum and colic angle is very narrow—it seemed possible that a volvulus might take place.

The patient left hospital in three weeks, and has been entirely free from symptoms up to the present date, March, 1930.

I have described a similar case also in an adult³—a female, age 57—which terminated less happily, and feel certain that had I then been familiar with this condition it might have ended differently.

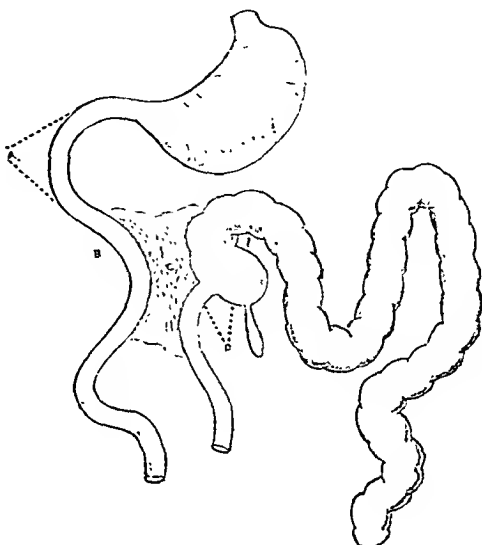


FIG. 102.—Diagram to show condition found at operation. A, Fixed duodenum; B, Site of junction of fixed duodenum with jejunum; C, Narrow attachment of mesentery of mid-gut loop. D, Floating cæcum and ascending colon.

REFERENCES.

- ¹ DOTT, NORMAN M., "Anomalies of Intestinal Rotation", *Brit. Jour. Surg.*, 1923, xi, 251.
- ² WAUGH, GEORGE, "The Morbid Consequences of a Mobile Cæcum and Ascending Colon", *Ibid.*, 1920, vii, 343.
- ³ GRANT, GEARY, "An Anomaly of Intestinal Rotation", *Surg. Gynecol. and Obst.*, 1928, xlvii, 133.

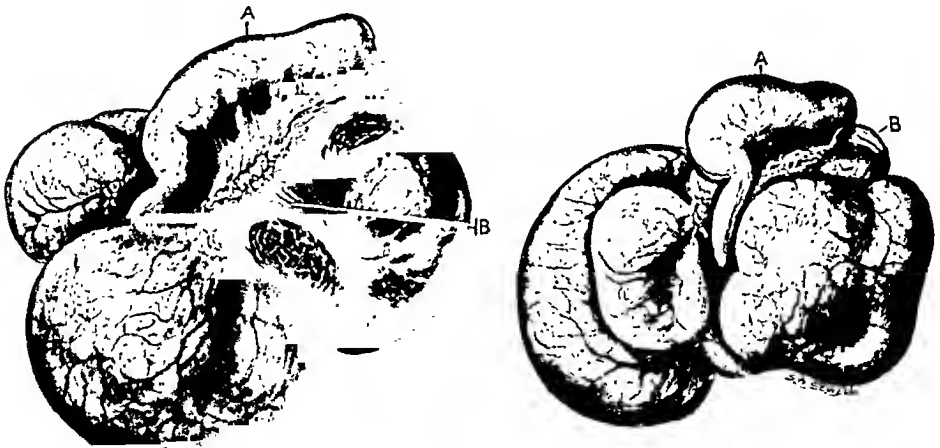
A RARE CASE OF INTESTINAL OBSTRUCTION.

By E. KENDALL,

RESIDENT MEDICAL OFFICER AT POPLAR HOSPITAL FOR ACCIDENTS, LONDON.

FOLLOWING so soon upon the paper by Mr. Arthur Evans in the *BRITISH JOURNAL OF SURGERY* in July, 1929, in which he deals with developmental enterogenous cysts, this rare case seems to be of interest.

HISTORY.—On July 14, 1929, J. A., a male, age 2 years 5 months, was sent to Poplar Hospital with a provisional diagnosis of acute appendicitis. On the evening of July 11 the child began to vomit and appeared listless. The next day the vomiting became more frequent and projectile in nature. Water only was given, and that was always vomited almost immediately. On the 13th he was seen by a doctor, who examined him and found the abdomen moving well but slightly distended. There was no rigidity. An enema was given and a good faecal result obtained. The child was examined



FIGS. 103, 104.—Appearance of cyst. A, Proximal end of small intestine; B, Distal end of small intestine.

again the next day and a provisional diagnosis of appendicitis was made and the child sent to us. The bowels had only been opened at the time of the enema. There was nothing in the previous history to draw attention to anything abnormal in the patient's abdomen.

ON ADMISSION.—The child looked very ill. His face was pale and drawn and the eyes were sunken. He vomited a thin brown fluid. The pulse was 136, the temperature 97°, and the tongue furred. Veins were very pronounced over the upper abdomen and chest. The upper abdomen was distended and a small-intestine pattern present. There was slight tenderness all over the abdomen, but not especially marked anywhere. Nothing abnormal was felt in the lower abdomen. A diagnosis of acute intestinal obstruction was made.

OPERATION.—A paramedian incision was made. The small intestine was very much distended. In the lower part of the abdomen was a large thin-walled cystic tumour about three inches in diameter causing a volvulus of the small intestine about one-third of the way down its length. The cyst was constricted in its middle by a thin purple band of small intestine which completely encircled it. The same intestine was twisted in a clockwise direction. On untwisting the volvulus the cyst was found to lie between the layers of mesentery, and seemed to be intimately connected with the wall of the jejunum (*Figs. 103, 104*). For about four and a half inches the bowel was flattened out in a ribbon-like band about half an inch across. The constricted intestine was dull and purple and would admit no contents from the proximal distended intestine. The cyst and volvulus were resected and an anastomosis was performed. The child died twelve hours afterwards.

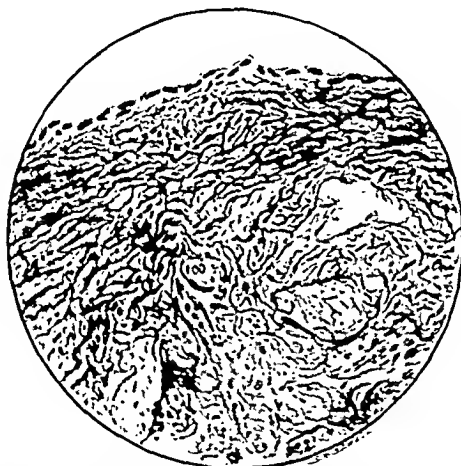


FIG. 105.—Wall of cyst showing endothelium, muscle, and elastic fibres. From a section stained with Weigert's iron-haematoxylin and van Gieson's mixture, and a section stained with Weigert's fuchsin. ($\times 235$.)

PATHOLOGICAL REPORT.—The cyst, a microscopical section of which is shown in *Fig. 105*, was examined by Dr. W. W. Woods at the London Hospital, and he reported as follows: "Cyst from mesentery with fibromuscular wall lined with endothelium, probably lymphatic." The cyst contained a serous, slightly straw-coloured fluid.

Mr. Arthur Evans quotes a very similar case reported by Robert T. Miller,¹ and definitely establishes that case to be an intermuscular enterogenous cyst projecting between the layers of the mesentery. The cyst mentioned in this paper, though very intimately connected with the small intestine, is lined by endothelium and thus not an enterogenous cyst. It is a strange coincidence that the cyst mentioned by Miller and the present cyst should bring about exactly the same pathological conditions and yet have such definitely different origins.

REFERENCE.

¹ *Johns Hopkins Hosp. Bull.*, No. 272, 316.

A CASE OF TRAUMATIC DIAPHRAGMATIC HERNIA.

By CYRIL POLSON,

LECTURER IN PATHOLOGY, UNIVERSITY OF LEEDS.

TRAUMATIC diaphragmatic herniæ, although unusual, are reported not infrequently, and this case presents certain features which make it worthy of record.

The patient, a male, age 43, was admitted to hospital on March 8, 1930, complaining of acute and severe abdominal pain. He vomited but once.

ON EXAMINATION.—An Argyll Robertson pupil and absence of the knee-jerks were noted. The patient was profoundly ill and died shortly after admission. Reference to his Army record shows that he was once wounded, on Nov. 4, 1917, when a shell burst amongst his platoon. He received an injury to his right thigh.

POST-MORTEM (March 10).—At autopsy extensive searring over the right hip with considerable deformity of the ilium was seen, but there were no scars elsewhere. When the sternum was removed it was at once seen that the left side of the thorax was occupied by the omentum, beneath which were the greater part of the transverse colon and several feet of the small intestine. The colon appeared to be healthy, but the small intestine had a deep purple colour owing to strangulation (*Figs. 106, 107*). There was about a pint of turbid fluid in the left pleural cavity. The left lung was completely collapsed, and the heart slightly displaced to the right.

Examination of the abdominal side of the diaphragm showed that the upper third of the stomach, with part of the small intestine and the greater part of the transverse colon, had passed through an aperture about three inches in diameter in the left dome of the diaphragm. This opening was surrounded by a ring of fibrous tissue. The small intestine lying in the abdomen also had a purple colour, but was obstructed to a less degree than that in the thorax. There was considerable traction on the mesentery, part of which had passed into the thorax. None of the other abdominal organs had entered the thoracic cavity.

Examination of the collapsed left lung revealed the presence of an irregular piece of steel measuring $\frac{1}{2}$ in. \times $\frac{5}{16}$ in. \times $\frac{1}{16}$ in., obviously a fragment of shell, in the lower lobe posteriorly. It was surrounded by a wall of fibrous tissue $\frac{1}{16}$ in. thick; otherwise there was little or no evidence of damage to the surrounding lung.

Commentary.—It is well recognized that there may be a considerable interval of time between the occurrence of a traumatic lesion of the diaphragm and the recognition of a subsequent hernia of abdominal contents. This man's wound was received in November, 1917, twelve and a half years before death, and it was only at autopsy that the hernia was discovered. In all probability it had existed from soon after the diaphragm was damaged, but there was nothing in the patient's history to indicate its presence until strangulation occurred.

The diagnosis presented considerable difficulty and was made only at autopsy. The occurrence of symptoms suggesting an 'acute abdomen' in the presence of evidence of tabes dorsalis pointed to a gastric crisis. The fact



FIG. 106.—Photograph of the body after preliminary opening of the thorax and abdomen, showing the viscera from the left side. The limits of the thoracic cavity are indicated by the cut ends of the costal cartilages. The middle third of the photograph is occupied by stomach and transverse colon, below and above the diaphragm (seen as a light triangular flap) respectively. Coils of strangulated small intestine lie beneath the transverse colon; the omentum is seen immediately to the right of the colon. Distended and strangulated small intestine is seen to the left of the stomach in the abdominal cavity. The stomach is dilated.

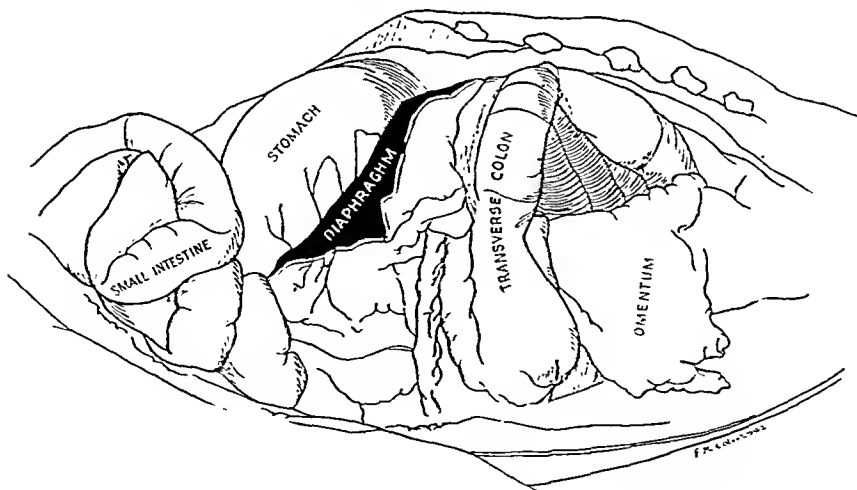


FIG. 107.—Diagram to indicate the disposition of the principal viscera.

that the patient vomited only once is particularly unusual, but is readily explained by the obstruction of the cardiac orifice caused by herniation of the stomach.

The external evidence of injury in the region of the right thigh, coupled with the discovery of a piece of shell in the left lung, permits the belief that the foreign body travelled upwards and inwards to the left from the thigh wound, there being no other scars on the body and no history of any other wounds. The rent in the diaphragm must have failed to close, herniation of part of the abdominal viscera occurring later, on a date which it is impossible to determine, since the first symptoms were those of the terminal acute strangulation.

Bryan (1921) has reviewed injuries of the diaphragm and describes the herniæ resulting therefrom. The condition of the viscera in this case agrees fairly closely with his description of the lesion.

I thank Professor Maxwell Telling for permission to record this case, and Miss Collinson for the drawing.

REFERENCE

BRYAN, C. W. G., *Brit. Jour. Surg.*, 1921-22, ix, 117.

A CASE OF SPINA BIFIDA OCCULTA.

By S. J. H. GRIFFITHS,

ASSISTANT SURGEON AT THE GENERAL HOSPITAL, BRISTOL.

G. P., a female domestic servant, age 23 years, was admitted to the Bristol General Hospital under the care of Mr. Hey Groves in June, 1928.

HISTORY.—The patient gave a history of occasional backache for as long as she could remember. She had been able to lead a normal life and do her work as a housemaid until nineteen months prior to her admission to hospital, when she fell from a window, ‘wrenching’ her back. She limped somewhat after the accident, but was able to continue at work for twelve months following the fall; then she complained of pain in the back, and found that she was gradually but steadily losing the use of the right leg. Apparently a diagnosis of tuberculous disease of the lumbar spine was made at this time, and she was sent to a sanatorium, where she was confined to bed for six months, unable even to sit up in bed owing to the pain in her back, and with increasing ‘paralysis’ of the right leg.

ON EXAMINATION.—The patient was found to be a well-nourished, healthy-looking girl, of normal development for her age. She was very miserable, and apparently in considerable pain when any attempt was made to move her lower limbs or back. Both legs were definitely spastic, and there was marked wasting of the whole of the right leg, in which the loss of power was extensive. The plantar reflex on the left side was a brisk flexor, on the right a feeble flexor. The knee-jerks were exaggerated on both sides. No sensory disturbances were found, nor was there any reaction of degeneration. The Wassermann reaction was negative. There was no deformity of the spine, but growing from the lumbar region was a thick tuft of dark, fine, silky hair, twelve to fourteen inches long.

X-ray examination of the dorsal and lumbar spine showed no abnormality.

A diagnosis was made of spina bifida occulta, and it was thought that the nerve symptoms were due to a fibrous arachnoiditis—that is to say, a fibrous band growing across the two halves of the laminae and crossing some part of the posterior part of the cord.

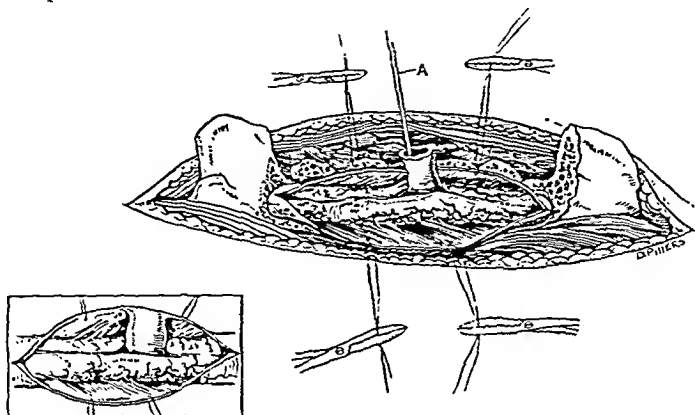


FIG. 108.—Spina bifida occulta. A, Probe in dural tube. Inset shows the condition of the spinal cord immediately the dura was opened.

OPERATION.—Laminectomy with exploration of the cord was thought advisable. This was performed and the laminae of D1 to L2 were removed. The area underlying the growth of hair was naevoid in nature and bled profusely, but the haemorrhage was easily controlled by adrenalin packs. Connected with the lamina of L1 a tubular prolongation of dura mater was noticed running down to a median opening in the lumbar enlargement of the cord. This was clearly displayed on splitting open the dura mater. It was found to consist of a tube, a quarter of an inch in diameter, running right across the spinal canal from the body in front to the lamina at the back. The spinal cord was punctured for its passage. On splitting open this tube it was found to contain a small spine of bone, growing backwards from the body in front. The cord was pulled towards the left and one of the posterior nerve-roots divided. The spine of bone occupying the tube was then cut away at its base and was removed with the tube of dura mater. (Figs. 108, 109.)

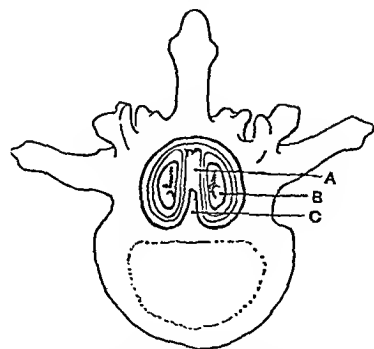


FIG. 109.—Transverse section of spine at seat of lesion. A, Dural tube; B, Bifid spinal cord; C, Bony spur.

SUBSEQUENT PROGRESS.—The immediate recovery from the operation was good, and there was no shock. The wound healed by first intention. There was an extensive loss of sensation in both lower limbs immediately following the operation, but this cleared up within a fortnight. The left leg returned to normal, and the right continued to show slow but steady improvement in power.

The main interest in this case seems to lie in the question whether it was an example of splitting of the spinal cord or of a bifid spinal cord—that is, whether the cord was just pierced by the prolongation of the tube of dura mater.

I can find only two cases quoted of alleged local doubling of the spinal cord—those quoted by Bruce¹ and by Purves-Stewart.² There seems to be no doubt that they were cases of local doubling of the spinal cord and not just a mere splitting. These conditions were discovered at autopsy, and it was possible to cut series of sections of the spinal cord to prove this point.

Mention of this peculiarity has been made in the "Report of the Committee on Spina Bifida".³ The specimen there alluded to is now in the Museum of the Royal College of Surgeons of England (No. 337, Teratology), and is described as "A spina bifida with a process of bone projecting from the junction of the bodies of two vertebræ backwards across the neural canal to the laminae, perforating the spinal cord." As it bears a close relation to the case just described, a fuller description is worth quoting:—

The specimen consists of the last two dorsal and the two upper lumbar vertebræ. The neural arches of the vertebræ are deficient, those of the eleventh dorsal and second lumbar are closed by fibrous tissue. Through the deficiency in the arch of the twelfth dorsal vertebra there protrudes the sac of a spina bifida. The deficiency in the neural arch of the first lumbar vertebra is closed by the expanded posterior end of an osseo-cartilaginous element, which crosses the vertebral canal from before backwards and perforates the spinal cord. This element abuts anteriorly against the posterior surfaces of the bodies of the twelfth dorsal and first lumbar vertebræ with the intervening fibro-cartilage. The division of the cord occurs asymmetrically; on the left side the anterior column is alone represented in the upper part of the division; the left lateral column is traceable for a short way on the right division. In its lower part the left division of the cord becomes nearly equal in size to the right, the lateral and posterior columns reappearing. The central canal is largely dilated above the point of division; the dilated canal traverses the upper part of the right division, the dilatation ceasing too in its lower half. Into the mouth of the sac there projects a diverticulum of the dilated right division of the cord. The roots of the last dorsal nerve arise, the anterior from the left division, the posterior from the right division of the cord; of the nerves below, both the roots arise from the reconstituted division of the cord of connective tissue.

It seems that in the case of our patient the symptoms were in no way due to any disease of the spine or the alleged accident, although it is true that the accident certainly seemed in her mind to be the starting-point of her symptoms. It seems clear that it was a case of spina bifida occulta, associated with local splitting of the spinal cord, producing symptoms well on in adult life. Operative treatment gave relief, for on discharge from hospital, eight months after the operation, the patient was able to stand and walk a short distance with the aid of crutches and a caliper splint on the right leg.

My thanks are due to Mr. Hey Groves for kindly allowing me to report this case; to Mr. Powell, of the Royal Society of Medicine, for his help in the research into the literature of this condition; and to Miss D. Pillers for the diagram of the operative findings.

REFERENCES.

¹ BRUCE, *Rev. Neurol. and Psychiat.*, 1905, iii.

² PURVES-STEWART, *Ibid.*, 1906.

³ *Trans. Clin. Soc. Lond.*, 1886.

REVIEWS AND NOTICES OF BOOKS.

Stone, and Calculous Disease of the Urinary Organs. By J. SWIFT JOLY, M.D. (Dub.). F.R.C.S., Surgeon to St. Peter's Hospital for Stone, etc. Crown 4to. Pp. 568 + xviii, with 189 illustrations in the text and 4 coloured plates. 1929. London: William Heinemann (Medical Books) Ltd. 45s. net.

*Omne tulit punctum qui miscuit utile dulci,
Lectorem delectando, pariterque monendo.*

WE do not apologize for making use of this somewhat well worn quotation, for we feel that these two lines of Horace are singularly applicable to the author of this monograph. The reader's first impression of this large volume of 568 pages may well be one of respectful wonder that any man could find so much to write on the subject of urinary stone; his second may be one of mild alarm lest the contents should prove to be dreadfully dry. After a careful perusal these impressions soon die away.

The book is long because Mr. Swift Joly has collected a vast amount of information pertaining to his subject, and also because his mind is so intensely inquisitive that he wants to know the why and wherefore of every phenomenon; it is hardly an exaggeration to say that he never makes a statement without discussing the reasons for it.

It certainly is not dry, though we confess that he has not a very light touch and that his paragraphs are often long, sometimes distressingly so. On the contrary, the reader becomes more and more fascinated as he realizes that this author is not merely a skilful surgeon but a man of much scientific and literary erudition. To support his theories and to emphasize his facts he calls in the aid of history, evolution, paleontology, chemistry, and physics, and he seems to be equally at home in each of them. Not only is his knowledge extensive, but he has the gift of presenting it to his readers in a form easily to be understood. The clearness of his exposition of such difficult questions as the nature of solutions, osmotic pressure, surface tension, and adsorption reminds us of the writing of the great Thomas Huxley.

The book starts with a brief résumé of the history of the surgery of stone, and it is admirably done; the author has collected a large number of facts, and they all point to the conclusion that in no other field does history so often repeat itself. We learn that in India suprapubic lithotomy was done in the time of Christ, that the superphysician, Hippocrates, knew all about the symptoms of stone in the bladder, and that Celsus wrote an excellent account of lateral lithotomy and of the use of catheters. After this we are hardly surprised to read that there existed at Cairo in the thirteenth century a hospital with separate wards for important diseases, staffed with male and female nurses and having an out-patient department: there seems to be no mention of any wireless installation, but there was the equivalent—professional story-tellers to comfort the sleepless.

Even the lady doctor was represented in the twelfth century in the person of Trotula: she wrote a book on genito-urinary disease. We wonder what the Church had to say on this matter: in those days she was a stern parent and disapproved of men's work being done by women, as witness the tragedy of Joan of Arc. We hardly raise our eyebrows when we hear that Morand used the Trendelenburg position some century and a half before the birth of its inventor, and that organotherapy was known to the ancients, for the testicles of goats were given in the treatment of impotence. Altogether a delightful chapter, with enough material for the elaboration of half a dozen lectures.

We consider that the third chapter is the most interesting in the book; in it the author discusses the composition and formation of calculi, their general characteristics, and the question of the etiology of stone. He classifies stone under three headings: (1) Those composed of substances normally found in the urine of man, such as uric acid and calcium oxalate; (2) Those formed from substances resulting from disordered metabolism, such as cystin; (3) Those formed from salts which appear in the urine as the result of bacterial decomposition, such as the triple phosphates. We think that this is the most scientific and helpful classification that has been advanced up to the present.

The author's theory of stone formation may be very briefly summarized as follows: all stones are formed of crystalloids and colloids, the latter representing the mortar which holds the bricks together. Human urine is a supersaturated solution in which the salts are held in solution by means of a colloid suspension; in the case of the passage of crystals in the urine such as is seen in the conditions known as uraturia, oxaluria, and phosphaturia, there is no excess of salts in the urine, but their precipitation is due to a deficiency in the colloids. It follows that the problem of the prevention of stone formation probably lies in the study of these colloids; in fact, the author goes so far as to suggest that, if a substance could be discovered that would remove the colloids without damaging the renal and bladder epithelium, the stone might fall apart into its constituent crystals, which might quite well be washed away by the stream of urine.

The discussion on the evolution of these colloids will delight all thoughtful surgeons; it is pointed out that fish and amphibia have no need to conserve water in their systems, and their urine is very dilute and has a very low specific gravity; indeed, it is probably excreted by a simple process of filtration, and the frog is said to pass its own volume of urine in twenty-four hours. Such a simple filtration would be highly inconvenient in man, for, if his urea were excreted in simple solution, it would entail the passage of from ten to twenty litres of urine a day. The author mentions the curious anomaly that, whilst fish get rid of their nitrogenous waste as urea, the birds and reptiles—though higher in the scale of evolution—have made an apparently retrograde step and have reverted to the habits of the invertebrates in their excretion of nitrogen as uric acid. Palaeontology seems to offer a reason for this, but it is a striking fact that uric acid should have twice been abandoned in the course of evolution for the more up-to-date urea, especially as the concentration of this throws a great strain on the renal epithelium.

The author's definition of a primary stone is that it is one formed entirely of substances, crystalloids and colloids, found in the urine, whilst a secondary stone requires the presence of a foreign colloid; that of a secondary stone is that it is developed on a preformed nucleus, whilst a primary stone has none.

The explanation of the concentric striation of stone is ingenious; the author assumes that the primary stone is a loose collection of crystals held together by the colloids normally present in the urine. This will have a rough surface from the projection of crystals, and the irritation of the renal epithelium by this will cause an exudation of blood or serum, with the result that the next layer contains a large proportion of colloids and relatively few crystals. The surface of the stone thus becomes smooth and the irritation and consequent exudation are lessened; then a second layer of crystals is deposited and the cycle starts again. This explanation is fully borne out by the study of the architecture of stones.

On the whole, the author considers that stone is, if not a deficiency disease, due to a poor and monotonous diet, and he thinks that the obvious decrease in the frequency of stone in the bladder is explained by the better conditions under which the poor live nowadays.

The chapters on renal and ureteral stone are most excellent: whether Mr. Swift Joly is dealing with the symptoms, the diagnosis, or the treatment of these conditions, he is always clear, and he invariably tries to find a reason for the statements he makes.

The chapter on stone in the bladder is a long one and covers nearly a hundred pages; in it are made two very interesting and important statements. In discussing the rare vesico-urethral stone, a bladder stone which has become partially

impacted in the prostatic urethra, the author points out that such stones not only give rise to severe backward pressure, but that the amount of damage done to the kidneys is difficult to estimate owing to the impossibility of passing a catheter beyond them, and he considers that, in such cases, the blood-urea test is the one substantial fact on which one can rely. The second point concerns the treatment of stone in the bladder complicated by diverticula. He says of these that, as long as the diverticulum is filled with the stone, the patient suffers from little or no residual urine; if the stone is removed without dealing with the diverticulum, the patient is henceforth unable to empty his bladder. Indeed, in one case he records, after removal of the stone the patient suffered from complete and permanent retention. The moral is sufficiently obvious, the diverticulum should be excised in every instance.

The two plates showing cystoscopic views of stones in the bladder are beautiful, as are all the illustrations in this book. The descriptions of the operations of litholapaxy and suprapubic cystotomy are models of clear and helpful writing, and we know of no author who gives a better account of the various mechanical procedures in the surgery of the kidney and bladder.

After reading this book very carefully, we do not hesitate to assert that it is one of the best monographs we have read on any subject; we feel sure that 'Swift Joly on Stone' will be the classical work on this subject for many years, and we venture to think that surgeons all over the world will feel deeply indebted to him; we believe that it must have taken him many years to write it, and, if he is not feeling exhausted by the task, we urge him to write another monograph on hydro-nephrosis; he seems to us to be the only author we know who would be likely to throw light on this obscure subject.

The Story of a Surgeon. By Sir JOHN BLAND-SUTTON, Bart., F.R.C.S., Consulting Surgeon, Middlesex Hospital. With a Preamble by RUDYARD KIPLING. Demy 8vo. Pp. 212 + xii. with 28 illustrations. 1930. London: Methuen & Co. Ltd. 12s. 6d. net.

It is a great tribute to the interest which the author has aroused in the minds of the public, by his personality and speaking, that this book should have received such a wonderful reception that for a time it was a veritable 'best seller'. We confess ourselves to feelings of elation and excitement on receiving and beginning the perusal of the book, but also we must admit that an almost proportionate feeling of disappointment followed the completion of its reading.

Of course it is full of interest from cover to cover—from the quaint piece of Kipling which introduces it, to the disjointed remarks about writing and illustrating books which finishes it. It gives a series of pictures of the author's life and thoughts in relation to various problems and activities of science, surgery, and religion. Travel, natural history, the Zoological Gardens, and the books he has written are the chief topics in addition to those of personal progress and interest in anatomy and surgery. There are many little verses interspersed among the narratives which are quaint if not beautiful. But whilst every page is interesting and extraordinarily characteristic of the author, yet the disappointment in the book as a whole remains, and is due to its disjointed character and its failure to give any sustained view of Bland-Sutton or his life.

A Shorter Surgery. A Practical Manual for Senior Students. By R. J. McNEILL, LovE, M.B., M.S. (Lond.), F.R.C.S., Assistant Surgeon, Metropolitan Hospital; Hunterian Professor R.C.S. Second edition. Demy 8vo. Pp. 371 + viii, with 74 illustrations, including 31 plates (one coloured). 1930. London: H. K. Lewis & Co. Ltd. 16s. net.

THE popularity of the first edition of this book has ensured a welcome for the second, and criticism may be deemed superfluous. Though enlarged, the book is still small

enough to be attractive; it is easy to read, and is well illustrated. It gives such an impression of completeness that many who are seeking for the easy road to knowledge feel that at last they have found it; yet they have nobody but themselves to blame for their unwarranted over-confidence, since in the preface—which, no doubt, they took to be the only useless page in the book—Mr. Love warns them that “it is intended that this volume should be used in conjunction with one of the many excellent surgical textbooks already existing”. In fact, it is offered to students as a ‘cram’ book, and those who want it in spite of the warning should be careful not to over-estimate its value.

It is also stated in the preface that it is “an attempt to crystallize the more important principles of surgery”. In this the author cannot be said to have succeeded, for the book deals not with principles but with details—hence its popularity. Many a candidate who makes the proud boast that he knows a ‘cram’ book by heart does not realize that the reason for failure in the examination is that, instead of building his knowledge upon fundamental principles, he has been memorizing a list of more or less isolated facts which, owing to the size of the book, cannot be exhaustive.

If a book is small enough to be learnt by heart—which implies that every statement in it is accepted without criticism—the author must realize that, although some dogmatism may be permitted in matters of opinion, strict accuracy must be observed in matters of fact. To state that radon loses half its radio-active properties within forty-eight hours; that the original cysts which Baker described were distended bursæ in connection with Charcot’s joints; and that the sequence of events in the production of Pott’s fracture is that the astragalus is forced against the external malleolus, which fractures, and the internal ligament is torn—is to delude the trustful student.

One of the chief causes of failure in examination papers is the candidate’s inability to express himself in language which conveys his meaning without ambiguity. A book designed to prepare students for examinations should be a model of clear writing, and the failure to fulfil this condition is the most serious defect in this one. The following may be mentioned as examples of many such misleading statements—that in gangrene the affected portion is separated from healthy tissue by a process of anæmic ulceration; that in carbuncles of the face infective thrombosis may spread along the facial vein to the pterygoid plexus and thence to the cavernous sinus; that the musculospiral nerve is commonly involved as a complication of fracture; that in carcinoma of the tongue pain may be referred to the ear along the auriculo-temporal nerve; that on examination of the swelling in the breast in a case of chronic mastitis “slight fixity to the skin may be detected”; and that on rectal examination in a case of intestinal obstruction by a gall-stone “a loop of ileum may be felt containing a gall-stone which has rolled over the brim of the pelvis into the pouch of Douglas”. The frequent repetition of ‘diverticulæ’ and ‘sequestræ’ becomes irritating even to the comparatively illiterate reader.

Although the book contains a great deal of information, the student is unable to discriminate between the facts which are important and those which are of less importance or even of questionable integrity—a criticism which applies more or less to all ‘cram’ books. If used as the author suggests, it may be of some value; but a glance at the paragraphs dealing with the treatment of acute osteomyelitis, acute infection of joints, and acute empyema will show how small is its value as a guide to the practice of surgery. Even in examinations the performance of gastro-enterostomy after suture of a perforated gastric ulcer, and the attempt to reduce a Colles’s fracture by grasping the hand as for a handshake, do not always meet with success.

With very few exceptions the illustrations are excellent, and the index is adequate. Though it should be possible to produce a book of this size to act as a scaffolding upon which the student could build his knowledge of surgery, the present volume cannot claim to have achieved this object—perhaps because it was never designed for such a purpose.

Surgical Diagnosis. By American authors. Edited by EVARTS AMBROSE GRAHAM, A.B., M.D., Bixby Professor of Surgery, School of Medicine, Washington University, St. Louis, etc. 9 $\frac{1}{2}$ " x 6". Vols. I and II. Vol. I. Pp. 919, with 508 illustrations. Vol. II. Pp. 871, with 326 illustrations. To be issued in three volumes and desk index volume. 1930. London and Philadelphia: W. B. Saunders Co. Per set £7 10s. net.

THE editor, in presenting this work to the public, raises a point of some practical importance. He mentions the very rapid developments in surgery which have occurred in the last quarter of a century. In commenting on this rapid development, and the removal by the use of anæsthetics of the fear of operations, he states that a new and rather unsuspected danger has arisen. By this he means that, owing to the very ease and safety with which operations can be done, there is a danger of unwise and unnecessary operations being performed. It appears also that in the United States of America, as in England, surgery is no longer concentrated in a comparatively small number of large towns, but is being practised in small towns, villages, and cottage hospitals in a manner which would have been impossible a generation ago. It might be said that the bulk of the surgery of the country is now in the hands of men who have not been trained to surgery, but who perform operations as a part of their general practice. For these reasons the editor considers that a work on surgical diagnosis should fill a useful place in the library of general practitioners, because he thinks—and there is very little doubt that he is correct—that many patients are operated upon after too little preliminary study. The book, then, is meant for surgeons and general practitioners, and not for students.

In it the names and aspects of disease commonly included in text-books are mentioned, but in addition the etiology and pathology are discussed, particular care being directed towards the method of clinical examination, though the methods of the laboratory are by no means neglected. Treatment, or rather the details of operative treatment, are not covered, because it is considered that there are many excellent books on operative surgery dealing with this side of the question.

The whole work is to be published in three volumes, together with a 'Desk Index Volume'. The first two volumes have now appeared. Volume I consists of 919 pages with 508 illustrations, and contains the following articles: Wounds; Infections; Post-operative Complications; Diseases of the Blood-vessels; Extremities, Bones and Cartilages, Muscles, Fascia, and Tendons; Diagnosis of Infections of the Hand; Diagnosis in Joint Disease and Injury; Diagnosis of Various Lesions of the Spine; Idiopathic Bone Fragility; Fractures and Dislocations of the Extremities.

Volume II consists of 871 pages with 326 illustrations, and contains articles on: Gynecological Diagnosis; the Skin and Subcutaneous Tissues; Diseases of the Face, Mouth, and Jaws; Diseases of the Neck; Diseases of the Thyroid and Parathyroid Glands; Surgical Diseases of the Stomach; Surgical Diseases of the Duodenum; Diseases of the Peritoneum; Diseases of the Appendix, Small Intestine, and Colon; Spleen; Hernia; Diagnosis of Acute Abdominal Emergencies. Both volumes are indexed.

The work is that of many contributors, forty-two in all, and therefore varies somewhat in quality; but on the whole we think the book is an extremely good one. A perusal of the article dealing with the subject in question cannot fail to be of value to a general practitioner who is about to operate on a patient.

Chirurgia clinica Polonica. Edited by A. JURAŚ (Poznań) and M. RUTKOWSKI (Kraków). With the collaboration of A. JIRÁSEK (Praha), L. KRYŚSKI (Warszawa), A. LEŚNIEWSKI (Warszawa), K. MICHEJNA (Wilno), T. OSTROWSKI (Lwów), Z. RADLINSKI (Warszawa), B. SAWICKI (Warszawa), H. SCHRAMM (Lwów), I. WIERZEJEWSKI (Poznań). Vol. I. Editio cum auxilio "Fundusz Kultury Narodowej". Royal 8vo. Pp. 424. Illustrated. 1929. Kraków: Gebethner & Wolff. Paris: Librairie franco-polonaise et étrangère.

THIS volume of Polish clinical surgery is a courageous attempt to make the work of surgeons in Poland accessible to those who are ignorant of the Slav languages. It contains five papers which have been already published in Poland, although there is no record of where they appeared.

Four of the papers have been translated into French and one into English. They form a substantial contribution to surgery and are well deserving of the wider publicity here given them. The first paper deals with the pathogenesis of infective osteitis, and is contributed by Dr. Stanislas Nowieki, Chief Assistant in the Cracow Surgical Clinic. His experiments were made on puppies and rabbits with *Staph. pyogenes aureus*, and were directed to show the effects on different parts of the bone, the cause of the necrosis, and the method by which nature deals with the dead bone. A good but not complete bibliography is appended and the paper is illustrated with twenty-six figures in the text.

The second paper deals with volvulus of the sigmoid, and is written by Dr. Jean Glatzel, Doctent in the Medical Faculty of the Jagellonne University of Cracow. Like the preceding article it is translated into French, and gives details of 154 cases with a mortality of 38 per cent. Dr. Glatzel recommends untwisting the volvulus at the earliest possible moment, followed by resection of the sigmoid and lateral anastomosis two or three weeks later. He states that such a resection has a mortality of 0 per cent. When the loop is gangrenous it should be excised at once and the intestine joined by circular enterorrhaphy. This paper is also accompanied by a bibliography of 167 references. Dr. J. Krotoski, Assistant in the Surgical Clinic of the University of Posnan, writes on the treatment of tuberculous disease of bones and joints by salts of gold. He states that he has obtained satisfactory results by intravenous injection of sanoerysin and of aurosane, and that the effects of the two drugs are identical. Details of seven cases are given. The paper is well illustrated with twenty-eight figures, and there is a bibliography.

Professor Arnold Jirásek, of the Prague University, Charles, supplies notes as to the surgical treatment of epilepsy, and finally Professor Anthony Jurasz, Director of the Surgical Clinic at the Posnan University, supplies a contribution in English on the operative treatment of Graves' disease. He believes that the best method consists in the subtotal excision of both lobes of the thyroid when all medical means have failed to effect a cure or ameliorate the condition.

It would be wise if the editors of future volumes would submit proofs of the articles to English, French, German, and Italian friends who would render them idiomatically. In the present volume some of the articles have been translated so literally that the sense is obscured.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Dudley Road Hospital, Birmingham. Second edition, revised and enlarged. Medium 8vo. Pp. 268 + xviii, with 306 illustrations, some of which are in colour. 1930. Bristol: John Wright & Sons Ltd. 21s. net.

THE first edition of this book was reviewed in Volume XV of this JOURNAL, published in January, 1928. The favourable opinion of the reviewer seems to have been shared by the medical profession, for the author has produced a second and enlarged edition with the idea of making the book useful to the advanced student and the general practitioner. We would like to repeat that this is an admirable book, dealing with methods of examination used in clinical surgery. There is a danger in making the book too big. If it is made much bigger, students will not read it, and that would be a pity.

Orthopädie im Kindesalter. By Hofrat Prof. Dr. HANS SPITZY (Vienna), with the collaboration of Geh. Hofrat Prof. Dr. FRITZ LANGE (Munich). Third edition, fully revised and enlarged. Crown 4to. Pp. 504 + vi, with 253 illustrations. 1930. Leipzig: F. C. W. Vogel. Paper covers, M. 45; bound, M. 50.

HANS SPITZY has so revised and enlarged his popular pre-war text-book in collaboration with Fritz Lange that for practical purposes the work is a new one. Its scope is deliberately confined to the orthopædic problems of childhood, and in this respect is a well-balanced exposition of Austrian and German practice. But as a work of

reference for the experienced surgeon the book is disappointing. Its perspective continues to be dominated by the older orthopædic surgery. Thus subjects like the spastic paralyses of childhood are considered at length, whilst much less attention is devoted to the prevention and treatment of the disabilities following common injuries, which form such a large proportion of the clinical material of the orthopædic surgeon to-day. Scoliosis, which in this country is gradually receding into the background, is also accorded a place of honour.

The style of the book is simple and attractive, and for a Teutonic monograph is unusually easy to follow. The illustrations are well chosen and informative. The reputation of the authors is a guarantee that the book is authoritative, and we must regard it as a useful supplement to the standard works on orthopædic surgery.

E. Stierlin's *Klinische Röntgendiagnostik des Verdauungskanal's*. By Dr. H. CHAOUL (Berlin), with a Foreword by FERDINAND SAUERBRUCH. Second edition, fully revised. Imperial Svo. Pp. 642 + ix, with 893 illustrations, most of which are reproductions of radiograms. 1928. Berlin: Julius Springer. Munich: J. F. Bergmann. Paper covers, RM. 84; bound, RM. 88.

THIS is one of the classical German text-books on the radiological diagnosis of diseases of the alimentary tract. There is a short chapter on technique which should prove of value to radiologists. In addition to the common methods of examination, the author describes the use of a small air-filled compression bag by means of which he obtains very satisfactory skiagrams of the mucous membrane relief. This is used especially for the diagnosis of gastritis.

The radiological findings in the various diseases of the alimentary tract are described in detail, also the appearances seen after operative procedures of various kinds have been undertaken. A short account of the normal appearances is also given. No doubt the short type of stomach is physiological for the Teutonic race, but it is to be feared that the author would condemn three-quarters of the English as gastropototics.

The descriptions in the text are orderly, clear, and concise, and the excellent quality of the reproductions makes the book of the greatest value to anyone wishing to study this subject. A very large bibliographical index is appended, and also an index to the book itself. No doubt the large number of illustrations accounts for the high price of the book. One warning only is necessary before buying this excellent work—do not buy an unbound copy, as it will soon fall to pieces; no doubt a bound one will be more lasting.

Radium and its Surgical Applications. By H. S. SOUTTAR, D.M., M.Ch. (Oxon.), F.R.C.S., Surgeon, London Hospital. Fcap 4to. Pp. 57 + vi. Illustrated. 1929. London: William Heinemann (Medical Books) Ltd. 7s. 6d. net.

THE author describes concisely the methods of radium therapy which he employs for the treatment of various lesions, and at a time when there are as many methods as there are radium surgeons, this type of book is useful, especially for those who would improve or change their own methods. It is to be regretted that no statistics are given, as it is by this means alone that any new form of therapy may be assessed; but from the prevailing optimism which pervades the book, it may be assumed that the author compares radium therapy very favourably with surgery.

This book should be useful to the general practitioner, who is to-day expected to be able to explain to his patients the relative merits of surgery and radium therapy; it is neither too long nor too complicated, and there is a very excellent and simple introduction to the physical aspects of this subject. If any criticism is to be made, it is that the difficulties of radium therapy have not been sufficiently emphasized, and this book may tempt general practitioners to do what should continue to be done in special institutions if further progress in the science is to be made.

Anæsthesia and Anæsthetics. By F. S. ROOP, M.B., B.S. (Durham), Anæsthetist to University College Hospital; and H. N. WEBBER, M.A., B.Ch. (Cantab.), Anæsthetist to University College Hospital. Demy 8vo. Pp. 292 + xi. Illustrated. London: Cassell & Co. Ltd. 14s. net.

This book contains in its somewhat limited compass a practical and readable account of the methods of general and local anæsthesia most commonly used in our English clinics at the present time. A welcome feature of the work is the good perspective in which the newer and older methods have been presented.

In Chapter 2 the respiratory aspects and difficulties of administration are discussed in a masterly manner, and this section of the book cannot fail to impress upon the reader the importance of close and uninterrupted attention on the part of the administrator. The chapters dealing with emergencies and special difficulties are examples of the valuable nature of the work. No anæsthetist with the contents of these chapters in his mind ought to find himself nonplussed in any contingency. Local and regional anæsthesia are described as fully as is consistent with the small size of the volume; in this, as in other parts of their subject, the authors have wisely economized space by avoiding theory and confining their remarks to clinical phenomena, with the result that they have produced a useful guide to the practice of anæsthesia.

The book is well printed, and the illustrations are nicely reproduced.

The Modern Surgical Treatment of Pulmonary Tuberculosis. By BERNARD HUDSON, M.A., M.D. (Cantab.), M.R.C.P. (Lond.), Medical Superintendent, Victoria Sanatorium, Davos-Platz. Modern Treatment Series (General editor: F. G. CROOKSHANK, M.D., F.R.C.P.). Crown 8vo. Pp. 128. 1930. London: Jonathan Cape. 5s. net.

THE author is to be congratulated on having done a very difficult task well. He has written a book dealing with the whole subject of the surgical treatment of pulmonary tuberculosis, compressing this into some 120 pages. The book has been planned, as the author states in his prefatory note, "to present the subject in a form which will be useful to the practising physician". With this object in view, Mr. Hudson has dealt principally with the indications and contra-indications, the advantages and disadvantages, the successes which can be looked for and the disappointments and dangers which may be met with. He has given a very clear guide to the relative merits of the different procedures, such as artificial pneumothorax, thoracoplasty, and evulsion of the phrenic nerve. At the same time he has given a concise, though naturally brief, description of the technique.

There are, as is to be expected, a few points with which others may not be in complete agreement, such as the statement on page 53 that the presence of bilateral lesions with active disease is a contra-indication for unilateral pneumothorax; also the recommendation to break down adhesions by comparatively high pressures. The opening sentence in Chapter 14, "The operation of thoracoplasty always produces considerable shock, which, curiously enough, is increased when a general anæsthetic is used", cannot be passed uncriticized, as it is so contrary to the experience of most physicians and surgeons.

Oleothorax, which is becoming of increasing importance, has scarcely had its fair share of space allotted to it. Olive oil, used in preference to paraffin, is not mentioned, nor is the risk of big reactions. In the chapter on phrenic evulsion (not avulsion) the indications "as laid down by Alexander" do not give a sufficient idea of the extent of the usefulness of this operation.

These minor blemishes do not, however, materially detract from the value of the book. The subject matter is there, and—what is equally important—it has been placed before the reader with a clarity and simplicity which is most pleasing. The book is well printed and well got up. It costs five shillings only, and is of generous value.

A Synopsis of Surgery. By ERNEST W. HEY GROVES, M.S., M.D., B.Sc. (Lond.), F.R.C.S., Surgeon to the Bristol General Hospital; Professor of Surgery at Bristol University, etc. Ninth edition. Crown 8vo. Pp. 676 + viii. Illustrated. 1930. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

THE need for the publication of a ninth edition of Professor Hey Groves's book proves its continued usefulness and acceptability. The text has been revised and brought up-to-date, and new matter, such as radium treatment for cancer and the Winnett-Orr treatment of compound fractures, added.

The Treatment of Varicose Veins by Intravenous Injections. By J. D. P. McLATCHIE, M.D., C.M., Physician, Western Skin Hospital. Crown 8vo. Pp. 51. 1928. London: William Heinemann (Medical Books) Ltd. 3s. 6d. net.

THIS is a short book describing clearly and simply the treatment of varicose veins by injections. There is nothing that is new or of particular note. The author gives first choice to sodium salicylate as the drug for injection, and claims good results, though no numerical record is given of results or of complications. An extensive bibliography of twelve pages is included.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Surgical Diseases of the Thyroid Gland. By E. M. EBERTS, M.D., Surgeon to the Montreal General Hospital; Associate Professor of Surgery, McGill University. With the assistance of R. R. FITZGERALD, M.D., and PHILIP G. SILVER, M.D. Crown 8vo. Pp. 238 + xii, with 48 illustrations. 1929. Philadelphia: Lea & Febiger. \$3.50 net.

Some Aspects of the Cancer Problem. An account of researches into the nature and control of malignant disease commenced in the University of Liverpool in 1905, and continued by the Liverpool Medical Research Organization (formerly the Liverpool Cancer Committee), together with some of the scientific papers that have been published. Edited by W. BLAIR BELL, B.S., M.D. (Lond.), F.R.C.S., Hon. F.A.C.S., Fellow of King's College, London; Director of the Liverpool Medical Research Organization, etc. Imperial 8vo. Pp. 543 + xiv, with 90 plates. 1930. London: Baillière, Tindall & Cox. 63s. net.

Chirurgie des Kindersalters. By Prof. Dr. R. DRACHTER, Leiter der chirurgischen Abteilung der Universitätskinderklinik, München; and Dr. J. R. GOSSMAN, Assistenzarzt der Abteilung. Third edition, fully revised and enlarged. Crown 4to. Pp. 1031 + xvi, with 714 illustrations. 1930. Leipzig: F. C. W. Vogel. Paper covers, M. 125; bound in half leather, M. 135.

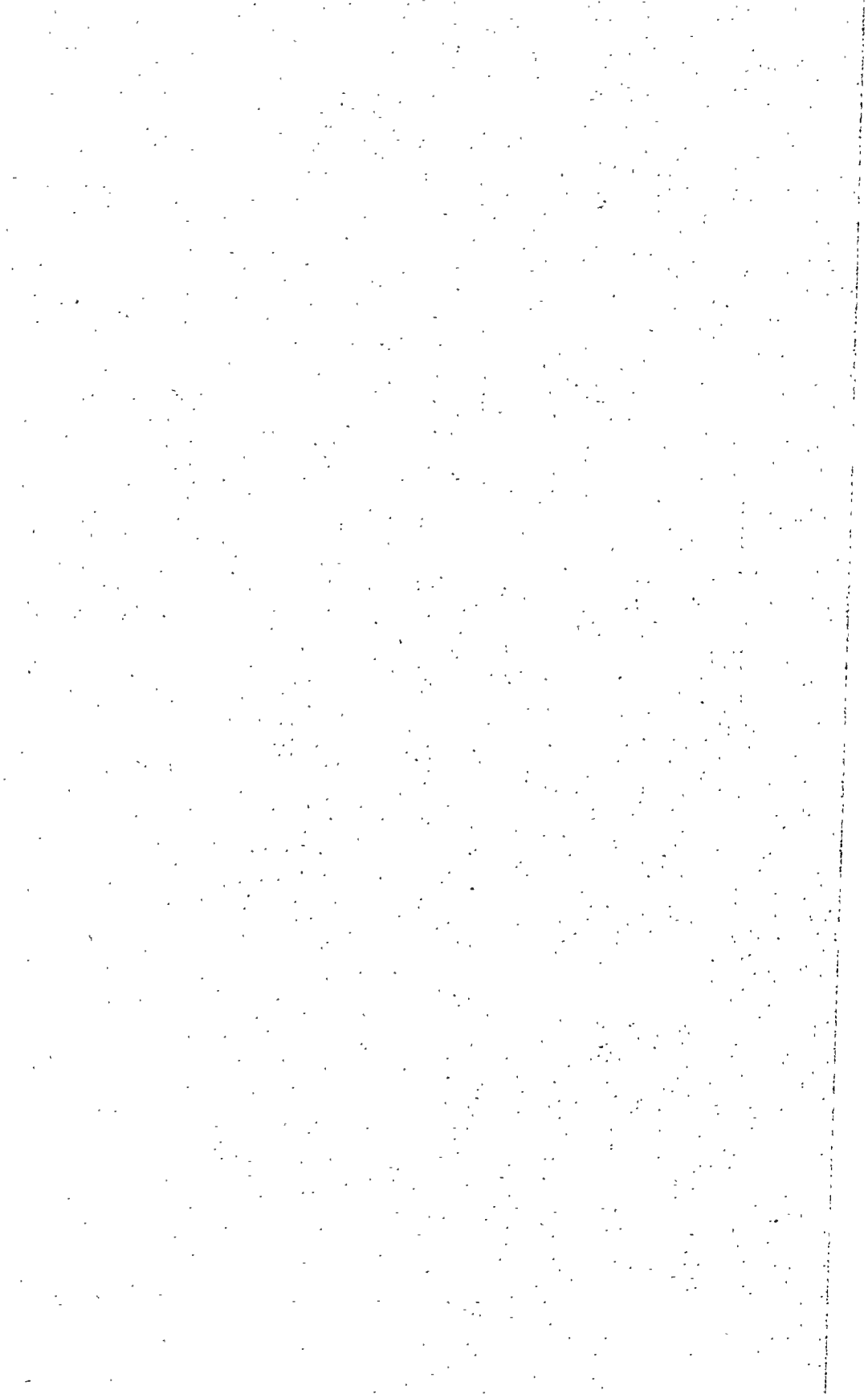
Radiologie clinique du Tube digestif. Published under the direction of PIERRE DUVAL, J.-Ch. ROUX, and H. BECLÈRE. II. Œsophagus, Intestine, Liver, etc., by J. GATELLIER, Professeur agrégé à la Faculté de Médecine de Paris; F. PORCHER, Chef de Laboratoire de Radiologie à la Faculté de Médecine de Paris. Fasc. 1 and 2. Royal 4to. Pp. 389, with 823 illustrations. 1930. Paris: Masson et Cie. Bound in two volumes for consignment abroad, Fr. 330.

Handbook on Tuberculosis. By B. S. KANGA, M.D., D.P.H., Medical Officer, Turner Dispensary, and Visiting Medical Officer, Turner Sanatorium (Bombay Municipality). Crown 8vo. Pp. 150 + viii. Illustrated. 1930. London: John Bale Sons & Danielsson. 5s. net.

The Clinical Pathology of Thoracic Puncture Fluids. By S. ROODHOUSE GLOYNE, M.D., D.P.H., Pathologist, City of London Hospital for Diseases of the Heart and Lungs, Victoria Park. Post 8vo. Pp. 83 + vii. Illustrated. 1930. London: John Bale Sons & Danielsson. 10s. 6d. net.

184 THE BRITISH JOURNAL OF SURGERY

- Injuries to Joints.** By Sir ROBERT JONES, Bart., K.B.E., C.B., Ch.M. (L'pool), F.R.C.S., (Eng., Ireland, and Edin.), F.A.C.S., Emeritus President, British Orthopaedic Association, etc. Third edition. Pott 8vo. Pp. 195. Illustrated. 1930. London: Humphrey Milford. 6s. net.
- Physikalisch-Chemische Probleme in der Chirurgie.** By Dr. C. HÄBLER, Privatdozent für Chirurgie in Würzburg. 9" x 6½". Pp. 274, with 62 illustrations. 1930. Berlin: Julius Springer. RM. 19.60.
- Die Chirurgie. A System of Surgery.** Edited by Profs. M. KIRSCHNER (Tübingen) and O. NORDMANN (Berlin). Fasc. 27 (Vol. III). Royal 8vo. Pp. 609-832, with 171 illustrations. 1930. Berlin and Vienna: Urban and Schwarzenberg. RM. 14.
- Minor Surgery.** By ARTHUR E. HERTZLER, M.D., Chief Surgeon, Halstead Hospital; and VICTOR E. CHESKY, M.D., Chief Resident Surgeon, Halstead Hospital. Second edition. Medium 8vo. Pp. 602, with 475 illustrations. 1930. London: Henry Kimpton. 42s. net.
- Sensation and the Sensory Pathway.** By JOHN S. B. STOPFORD, M.D., F.R.S., Professor of Anatomy, University of Manchester. Demy 8vo. Pp. 148 + xii. Illustrated. 1930. London: Longmans, Green & Co. Ltd. 7s. 6d. net.
- Guy's Hospital Reports.** Edited by ARTHUR F. HURST, M.D. April, 1930. Vol. LXXX (Vol. X, Fourth Series), No. 2. Royal 8vo. Pp. 127-252. Illustrated. 1930. London: Lancet Ltd. Annual subscription £2 2s. 0d. net, or 12s. 6d. net per issue.
- Congenital Club-Foot (Talipes Equinovarus).** By E. P. BROCKMAN, M.Chir., F.R.C.S., Orthopaedic Surgeon, Westminster Hospital, etc. Demy 8vo. Pp. 110 + viii, with 92 illustrations. 1930. Bristol: John Wright & Sons Ltd. 10s. 6d. net.
- L'Ostéo-synthèse métallique dans les Fractures diaphysaires. Etude expérimentale et anatomo-pathologique. Applications pratiques à la Chirurgie humaine.** By ROBERT FRANTZ, Ancien Interne des Hôpitaux de Paris. With a Preface by Professeur BERNARD CUNéo. Royal 8vo. Pp. 190. Illustrated. 1929. Paris: Masson et Cie. Fr. 35.
- L'Arthrite chronique de la Hanche.** By L. DUVERNAY, Ancien Interne des Hôpitaux de Lyon. Medium 8vo. Pp. 145. Illustrated. 1930. Paris: Masson et Cie. Fr. 40.
- Tecnico radiodiagnostica.** By Dott. GINO LASEHI, Aiuto dell' Instituto di Radiologia della R. Università Bologna. With a Preface by Prof. G. G. PALNIEIRI. 10" x 7". Pp. 288 + viii. Illustrated. 1930. Bologna: Licinio Cappelli.
- Die Knochenbruchbehandlung mit Drahtzügen.** By Prof. Dr. RUDOLF KLAPP, Direktor der chirurgischen Universitätsklinik, Marburg a.d. Lahn.; and Dr. WERNER BLOCK, Chefarzt der chirurgisch-gynäkologischen Abteilung am Marienhospital, Witten-Ruhr. 10" x 7". Pp. 294. Illustrated. 1930. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 25; bound, RM. 28.
- Report on Fifth International Congress of Military Medicine and Pharmacy, London, England, May, 1929.** By WILLIAM SEAMAN BAINBRIDGE, Commander, M.C.F., United States Naval Reserve; Member of Permanent Committee; Delegate from the United States. Medium 8vo. Pp. 154. Illustrated. 1930. Menasha, Wisconsin: George Banta Publishing Company.



THE BRITISH JOURNAL OF SURGERY

VOL. XVIII.

OCTOBER, 1930.

No. 70.

SOME BYGONE OPERATIONS IN SURGERY.

By SIR D'ARCY POWER, K.B.E., LONDON.

II. CUTTING FOR THE STONE (*continued*).

FRÈRE JACQUES' OPERATION.

CUTTING on the gripe and the Marian operation held their own throughout Europe until Frère Jacques, seemingly by accident, stumbled upon a better method, which—improved by Rau in Holland and by Cheselden in England—became lateral lithotomy as it was known and practised by the last generation of surgeons throughout the civilized world.

Frère Jacques, born in 1651, as Jacques Baulot, of poor parents in Franche-Comté, served as a trooper in a French cavalry regiment from 1657 to 1672. Released from service he acted as servant to Pauloni, a strolling Italian lithotomist and curer of ruptures, with whom he wandered for six or seven years through France and Italy. He adopted a semi-religious habit and began to call himself Frère Jacques in 1688, but it is doubtful whether he ever took any vows, though he may perhaps have become a lay-brother of the Order of Saint Francis. He did not assume a full religious habit, and is always represented as wearing an enormous hat, a relic perhaps of his strolling years.

In 1695 he made friends at Besançon, and two years later he was in Paris with a commendatory letter which led the surgeons at the Hôtel-Dieu to put his skill to the test by ordering him to extract a stone from a dead body. Jean Méry, a skilled anatomist, Surgeon to the Hôtel-Dieu, watched the operation, and afterwards made a careful dissection of the parts. He reported that the incision had passed between the cretor penis and the accelerator urinae without wounding either muscle; that the whole thickness of the prostate had been divided in the deeper part of the wound in a postero-lateral direction, and that the neck of the bladder as well as a portion of the wall beyond the prostate had been cleanly divided. Méry appreciated the value of the operation, for he pointed out that no important blood-vessel was involved, that greater space was given for the extraction of the stone, and that the tearing and bruising unavoidable in the Marian method was almost entirely avoided.

The results obtained by Frère Jacques at this time proved to be unsatisfactory. He cut 60 patients and 25 died, whilst the Paris surgeons, using the older method to which they were accustomed, only lost 3 out of 22. Frère Jacques was therefore discredited and returned for a year to his wandering life, in the course of which he visited Holland, where in 1699 he was so successful that J. J. Rau (b. 1668), a former pupil of Méry in Paris, who had been appointed the operating surgeon at the Amsterdam General Hospital, copied his methods with much success. In 1700 Jacques was recalled to Versailles, and G. C. Fagon (b. 1638), surgeon to Louis XIV, had him properly instructed in the anatomy of the parts on which he operated. Frère Jacques profited by the instruction, modified his operation, and practised with such success in Holland that the magistrates at Amsterdam presented him in 1704 with his portrait and a set of gold sounds. The portrait was painted by Poel and was engraved by Schenk.

Frère Jacques died in his native village on Dec. 7, 1719. He appears to have been a good Catholic and abounding in charity. He frequently operated upon the poor without fee, and is said to have melted down the gold sounds and distributed the proceeds amongst them. His name is connected with two distinct operations. The first one, learnt perhaps from his master the itinerant lithotomist, resembled that recommended by Pierre Franco in 1556 which had never come into general use; the second operation he evolved after Fagon had taught him some anatomy.

In the first operation a staff without a groove was introduced into the bladder, and an incision with a long dagger-shaped knife was made by the side of the left ischium. The incision was carried obliquely upwards, and divided everything between the tuber ischii and the staff until the wound was large enough to admit a finger into the bladder. An instrument which he called his 'conductor' was then passed along his finger, and along the conductor he passed a pair of forceps. Having introduced the forceps he withdrew the conductor, and as soon as the stone was found he withdrew the staff and then removed the stone by means of the forceps.

In the second method the round staff was replaced by one with a groove, and an ordinary scalpel was used instead of the long dagger-shaped knife. The position of the skin incision was the same as before, but the deeper incision was quite different; instead of passing the knife through the cellular tissues surrounding the bladder and cutting its posterior wall, the neck of the bladder and the prostate were divided by passing the knife along the groove in the staff. The rectum and the pudic artery were thus less likely to be injured. The operation proved so satisfactory that Frère Jacques cut 38 patients in succession at Versailles without a single death.

Rau watched Jacques operate, learnt the secret of the second improved method, practised it with success, but, to his lasting shame, allowed his colleagues and pupils to believe that he still divided the posterior wall of the bladder. Wounds of the rectum, extensive extravasation, and sloughing of the soft cellular tissues therefore continued to be the rule until Cheselden elaborated a still more satisfactory operation.



THE PATHOLOGY OF CONGENITAL TORTICOLLIS.

By D. STEWART MIDDLETON,

ASSISTANT SURGEON AT THE HOSPITAL FOR SICK CHILDREN, EDINBURGH.

AN explanation of the sequence of events leading up to congenital torticollis which carries with it even the least conviction has for many years been lacking in text-books of surgery. The present paper is based upon a clinical and pathological survey of 90 cases of torticollis and sternomastoid tumour which have come under my personal observation during the last few years. In addition, experimental work bearing upon the subject has been carried out in the Surgical Research Laboratory of the University of Edinburgh by permission of Professor D. P. D. Wilkie.

Any attempt to clarify the question of torticollis must deal with the following points: (I) The previous theories which have been advanced to explain the facts, and a short discussion of the weak points of such theories. (II) The relationship, if any, between congenital torticollis and the so-called 'sternomastoid tumour' of infancy: the morbid anatomy of the muscle in sternomastoid tumour and torticollis. (III) The etiology of the sternomastoid tumour: experimental reproduction of similar changes in muscle in animals. (IV) The etiology of various changes in the skeleton associated with the presence of torticollis.

I. THE OLDER THEORIES OF THE ETIOLOGY OF STERNOMASTOID TUMOUR AND TORTICOLLIS.

1. **The Hereditary Theory.**—This theory, supported by the observations of Jeannel, Pfeiffer, Fisher, and Joachimstal,^{4, 6} cannot be maintained. Cases of torticollis occurring in parent and offspring are so infrequent as to render it certain that they are merely the result of coincidence.

2. **The 'Congenital' Theory.**—The view that the contracture, like club-foot or hare-lip, is due to some abnormality in the development of the foetus, or to some abnormal pressure on the foetus by the walls of the uterus, might be held to account for those rare cases where an infant is born with a fully developed torticollis. In the vast majority of cases, however, the wry-neck does not appear till months or years after birth. Also it would be difficult to explain by this theory why the contracture should be completely limited to the sternomastoid muscle in all cases except old-standing adult ones where secondary adaptive changes in surrounding muscles and fasciæ take place. Finally, a congenital theory cannot account for the fibrosis present in the contracted muscle.

3. **The Theory of Birth Injury.**—Stromeyer, who formulated this theory, holds that the sternomastoid muscle is ruptured during labour, giving rise

to the 'sternomastoid tumour', which is in reality a hæmatoma, and that the contracture following the organization of this hæmatoma is responsible for the wry-neck. This is the theory which commonly receives support to-day.

As we shall show later, this theory contains a grain of truth in that it realizes that there is an intimate relationship between the presence of a sternomastoid tumour and the subsequent development of a wry-neck, and also that both these conditions have some connection with long and difficult labours. On the other hand, the theory errs in attributing the sternomastoid tumour to the presence of a hæmatoma following rupture of the muscle during labour. At no time does the swelling show any of the characteristics of a hæmatoma, being from its inception firm, hard, and cartilaginous to touch, and never fluctuating or showing any sign of bruising. It does not appear immediately after birth. It affects in most cases the greater part of the muscle instead of a localized portion of it, and on microscopical examination it shows no appearance which can in any way suggest a hæmatoma. Finally, true hæmatomata produced experimentally by tearing a muscle leave no trace of contracture behind them and in no way resemble sternomastoid tumours (Witzel).¹³

4. The Theory of Infective Myositis following upon Contusion of the Sternomastoid Muscle at Birth.—This was based upon the microscopical appearances of the excised muscle. It has been shown by Kempf,⁷ however, that there is no foundation for the belief that there is any infection present. He comes to the conclusion that the microscopical appearances seen are those of the repair of a hæmorrhagic infarct, a statement which is, as we shall see later, entirely true.

5. The Nervous Theory.—Few authorities support this theory. Golding Bird incriminated a cerebral lesion in intra-uterine life, while Gallavardin and Savy³ observed atrophy of the anterior-horn cells in the cervical cord in an adult case of torticollis. It is probable that these changes are secondary to the destruction of muscle, and are analogous to the atrophy of anterior-horn cells which follows upon the amputation of a limb.

6. The Ischæmic Theory.—First suggested by Mickuliez, this view was supported when Nové-Josserand and Viannay¹⁰ undertook a detailed investigation of the circulatory system of the sternomastoid muscle, and showed the possibility of interference with the blood-supply of the muscle taking place during labour.

This theory marks the most modern and most accurate work on the subject, and, as we hope to show, it comes very close to the truth. It recognizes that the muscular fibrosis seen in torticollis is essentially a similar process to that seen in Volkmann's ischæmic contracture of the flexor muscles of the forearm. It errs in adopting too whole-heartedly the belief which was then (1906) widely accepted, that Volkmann's contracture results from arterial deprivation to the flexor muscles, a belief which has always been and remains to-day completely unsupported by any sound evidence, clinical, pathological, or experimental. As we shall see later, such fibrosis in muscle can only result from venous obstruction, producing, in fact, the true 'hæmorrhagic infarct' described by Kempf.

II. RELATIONSHIP BETWEEN THE STERNOMASTOID TUMOUR AND TORTICOLLIS: MORBID ANATOMY.

It is commonly stated in text-books that though torticollis, when it appears early in life, may follow directly upon the absorption of a sternomastoid tumour, yet the majority of cases appearing later in childhood have not been in any way connected with such a condition. Both types of torticollis present the same clinical and pathological appearances, and it would therefore appear rational to expect that all cases are attributable to a common cause.

Let us consider the main facts with regard to sternomastoid tumour and torticollis.

The Sternomastoid Tumour of Infancy.—The tumour appears usually from one to two weeks after birth, the commonest time being about ten days. It is, so far as my observations go, never present at birth. It is first noticed as a spindle-shaped swelling occupying the position of one sternomastoid muscle. Occasionally it affects only the sternal head of the muscle, but very frequently both sternal and clavicular heads are thickened. The small uppermost portion of the muscle close to the mastoid attachment is, however, seldom if ever affected. The enlarged muscle is hard to the touch, and gives a typical 'cartilaginous' sensation to the examining fingers which I have never felt elsewhere apart from the experimentally produced tumour which will be described later.



FIG. 110.—I. H., male, age 6 weeks. Large sternomastoid tumour. Excised in part and sectioned to show fibrous-tissue replacement of muscle.

If a child suffering from such a tumour is kept under observation, it is found that the tumour remains more or less stationary for from two to three months, and then is gradually absorbed, disappearing in from four to six months after birth.

It is not uncommon for such a child to exhibit a mild degree of torticollis while the tumour is at its height, but this is transient and is of the nature of an acute torticollis. As the tumour is tender to touch, the child holds the head to one side in order to relax the affected muscle as far as possible. On the other hand, in some cases with a well-marked tumour, a true torticollis commences at about four months of age when the tumour is diminishing in size, the head being apparently drawn over to one side coincidently with the absorption of the tumour.

Morbid Anatomy.—If the sternomastoid tumour is excised and examined, it appears to the naked eye to be composed entirely of glistening fibrous tissue, and presents an appearance not unlike a soft fibroma on section (*Fig. 110*). On microscopical examination (*Fig. 111*) it is found to consist of young and cellular fibrous tissue containing here and there the remnants of the original muscle fibres. Many of these fibres show an absence of nuclei, vacuolation, and are undergoing degeneration.

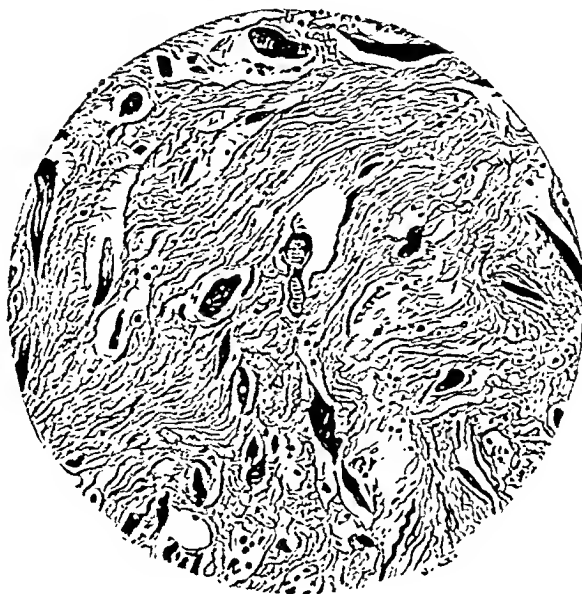


FIG. 111.—L. M., female, age 4½ months. Large sternomastoid tumour, with severe torticollis developing as resolution took place. The microscopical section is from the sternal head, and is stained by Azan's method. Cellular fibrous tissue and remains of muscle fibres are seen.



FIG. 112.—A. M., male, age 8 years. Torticollis. Deformity first noticed at 2 years of age. Parent could not recollect a tumour. Microscopical section from sternal head shows adult fibrous tissue with atrophic but viable muscle fibres.

Torticollis.—

Morbid Anatomy.—If the contracted sternal or clavicular head from a case of fully developed torticollis in a child of, say, 8 years of age is excised and examined (*Fig. 112*), the microscopical picture can only be interpreted as representing the terminal stage of a sternomastoid tumour. No degenerating muscle or young fibrous tissue is observed, but instead swathes of adult non-cellular fibrous tissue are seen, with, scattered amongst it, collections of muscle fibres which, though smaller than normal and varying somewhat in size and outline, are living healthy fibres and bear none of the stigmata of degeneration.

The microscopical appearances in themselves form convincing evidence that torticollis represents the end-result of a sternomastoid tumour; and, on investigating 36 cases of torticollis, it was found that 25 gave a definite history of a sternomastoid tumour shortly after birth which underwent absorption and was followed at varying periods up to some five or six years later by the development of torticollis. The cases in which no history of a tumour was obtained were mainly in older children, and are in all probability accounted for by changes in guardianship or the failure of a parent's memory to recall a trifling ailment of a passing character affecting one child of a family some six or ten years before. It is, in fact, an occasional experience in the out-patient department to discover a sternomastoid tumour which has remained unnoticed in a stout baby.

Reason for Late Development.—One point remains to be dealt with at this stage. How is it, if torticollis results from the fibrosis consequent upon the absorption of the sternomastoid tumour, that the wry-neck may not be noticed for years afterwards, in the majority of cases appearing about the age of 4? A little thought will make this clear. The neck of an infant is short, and it is not until about the age of 4 that the individual commences to grow from the proportions of a 'child' into those of the 'small boy or girl'. The whole bodily type of a child changes at this period. Prominent among the changes which occur is an increased growth in height, and this is especially marked in the relative increase in length of the neck, which assumes almost adult proportions. Suppose a child at this age to have suffered during infancy from a sternomastoid tumour which has become absorbed and left behind it a degree of residual fibrosis which was not sufficient to cause the immediate development of a torticollis. Such a muscle is crippled as regards the function of growth in comparison to its normal neighbour on the opposite side. The muscle substance on the affected side is partly replaced by an abnormal tissue which will not elongate with growth to the same extent as the normal muscle. When the growth period arrives, therefore, the fibrosed muscle lags behind in its elongation and becomes relatively shortened, producing the classical deformity.

III. ETIOLOGY OF THE STERNOMASTOID TUMOUR: EXPERIMENTAL INVESTIGATIONS.

We have already seen that the often-quoted theory that the tumour is a hæmatoma following upon rupture of the sternomastoid muscle during labour is erroneous, and that the ischæmic theory of Nové-Josserand and

Viannay errs in attributing the condition to arterial deprivation. Kempf's statement that the microscopical appearances are those of an organizing hæmorrhagic infarct comes very close to the truth.

Brooks,¹ of Chicago, has in recent years performed an interesting series of experiments upon isolated muscles in animals, and has established the following facts:—

1. Ligature of the artery supplying a muscle can produce atrophy of the muscle, but such atrophy is not accompanied by a massive formation of fibrous tissue. This fact is borne out by clinical observation. Ligature of the femoral artery, for instance, if it does not determine complete gangrene of the limb, causes marked muscular atrophy, but this atrophy is never accompanied by fibrosis and contracture in the affected muscles.

2. Ligature of the artery and vein together produces results which are essentially similar to those following ligature of the artery alone.

3. Ligature of the vein draining blood from the muscle alone, leaving the arterial supply untouched, results in a remarkable series of changes culminating in death of muscle fibres and a massive fibrous-tissue replacement of the whole muscle.

I have repeated these experiments, using some thirty dogs, and have found the observations of Brooks to be entirely accurate. I propose now to enter into a rather more detailed description of the changes following the operation of venous ligation in the isolated muscle of the dog.

Experimental Technique.—In all cases the sartorius muscle of the dog was used, as it is easy of access, and possesses a single vasculo-nervous bundle entering it at its proximal extremity. Under ether anæsthesia, and with surgical aseptic precautions, the muscle is exposed by a long incision down the front of the thigh. The muscle is separated from its sheath from origin to insertion save for the point at the medial aspect close to the proximal attachment where the vasculo-nervous bundle enters. This bundle is carefully dissected in order to expose its component parts. The nerve-supply to the muscle and the artery are carefully preserved, while the two venæ comites returning the blood are divided between ligatures. The operation is completed by tying a ligature around the two attachments of the muscle in order to interrupt anastomotic channels which may be present, especially at the proximal end. Immediately after this operation the muscle can be seen to become cyanotic in colour and the veins in the muscle become turgid. The fascial sheath and the skin incision are then closed with interrupted sutures.

Changes in Muscle following Venous Ligation.—Little change can be made out on palpation of the muscle for a week or ten days after operation, but after this period the muscle is felt to be hard and cartilaginous to touch. It conveys to the examining fingers a sensation which is exactly similar to that of the sternomastoid tumour in the child, and it will be noticed that this stage appears at approximately the same interval after operation in the dog as the tumour does after birth in the child.

A series of dogs subjected to this operation of venous ligation were killed at intervals up to six weeks afterwards and the muscles excised and examined. The accompanying illustrations (*Figs. 113-116*) show the naked-eye and microscopical appearances of the muscles.



FIG. 113.—Dog No. 13. Sartorius muscle two days after venous ligation as compared with the normal muscle from the opposite limb (left). Intense oedema and cyanosis.

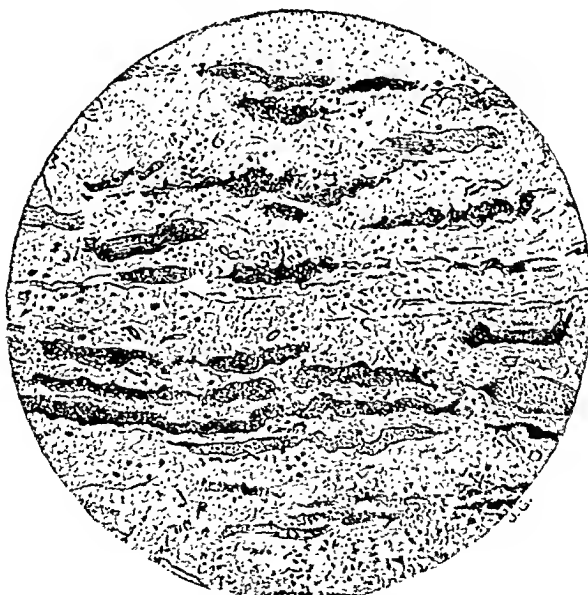


FIG. 114.—Dog No. 13. Two days after venous ligation. Microscopical section, stained hæmatoxylin and eosin, showing initial muscular degeneration and early invasion of the oedematous spaces by leucocytes.

Two days after operation the muscle presents a striking appearance. It is greatly enlarged, soft to touch, and dark blue, almost black, and necrotic in appearance as compared with the normal muscle from the opposite limb (*Fig. 113*). On microscopical examination (*Fig. 114*) the muscle fibres are seen to be separated by lakes of œdematous fluid, in which large numbers of polymorphonuclear leucocytes can be seen. The fibres are undergoing degeneration. It is to be noted here that all the experimental wounds healed cleanly by first intention, and in no case was any sign of suppuration evident.

One week after operation the naked-eye appearances are much the same, but on microscopical examination the leucocytic invasion is seen to be rather more intense.

Two weeks after operation the muscle is seen by the naked eye to be undergoing commencing fibrosis.

Eight weeks after ligation the muscle is seen macroscopically to be converted into solid fibrous tissue (*Fig. 115*). If the appearance of this muscle is compared with that of a sternomastoid tumour removed from a child six weeks after birth (*see Fig. 110*), the resemblance of the two tissues is striking. Microscopically, at this stage, young cellular fibrous tissue is seen to have replaced the



FIG. 115.—Dog No. 1. Sartorius muscle eight weeks after venous ligation. Almost complete replacement by fibrous tissue. This represents the experimental reproduction of the 'sternomastoid tumour' (*Fig. 110*).

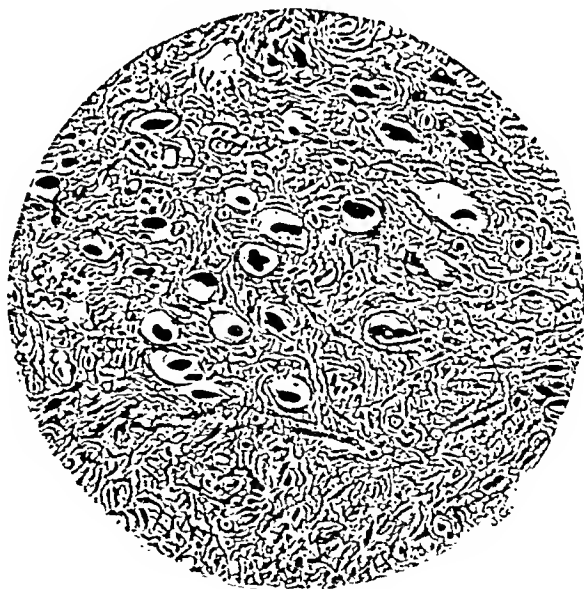


FIG. 116.—Dog No. 1. Microscopical section of sartorius muscle eight weeks after venous ligation, stained by Azan's method. Young cellular connective tissue and muscular remnants are to be seen. Compare with *Fig. 111*.

œdema of the earlier stages. The muscular tissue has largely disappeared, but amongst the fibrous tissue there can be seen remnants of more or less atrophic and irregular muscle fibres (*Fig. 116*). This section may be

compared with a corresponding microscopical section of a sternomastoid tumour (*see Fig. 111*), which shows a pathological appearance essentially similar.

Finally, if the figure illustrating the microscopical appearances of the fibrosed muscle from an 8-year-old child the subject of torticollis is glanced at (*see Fig. 112*), it will be clear that the muscle from torticollis represents the end-result of the sternomastoid tumour, adult non-cellular fibrous tissue being now in evidence.

These observations strongly support the view that sternomastoid tumour and consequently congenital torticollis result from an acute venous obstruction of the muscle taking place during birth.

It is of interest to notice that, as *Fig. 117* shows, the fibroblasts responsible for laying down the fibrous tissue are derived from the proliferation of the endothelium lining the smaller blood-vessels in the muscle.



FIG. 117.—Microscopical section from experimentally produced tumour of two weeks' standing, to illustrate the endothelial reaction which results in the budding off of numerous fibroblasts from the smaller blood-vessels.

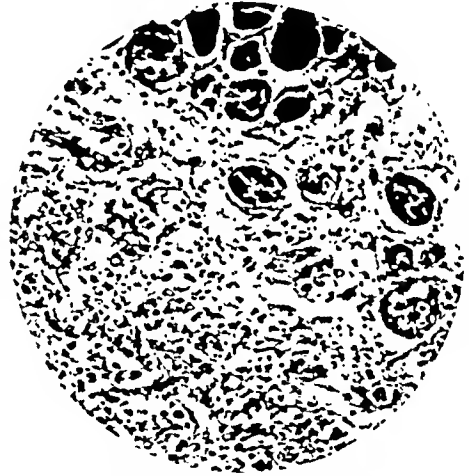


FIG. 118.—Microscopical section from experimentally produced tumour of ten days' standing. Stages in process of gradual disappearance of muscle fibres by autolysis can be seen.

It should be clearly realized that the death of the muscle, *per se*, is not the factor which determines the fibrous-tissue formation. It would appear that when a muscle fibre dies it undergoes autolysis, breaking up into a mass of granules which form as it were a 'shadow' of the original muscle fibre which gradually disappears (*Fig. 118*). Phagocytosis does not appear to be concerned in the removal of the products of autolysis. If the fibre dies of ischæmia consequent upon cutting off the arterial supply or the arterial supply and venous return together, so that no œdema results, no marked degree of fibrosis follows; but if acute venous obstruction is present along with a free arterial supply, the disappearing fibres, lying in œdematous spaces, are replaced by masses of fibrous tissue laid down in the œdematous fluid. In fact, the œdema appears to be the essential factor in causing the fibrosis.

Production of Venous Obstruction in Labour.—It is difficult at first sight to explain the mechanism by which such a venous obstruction can occur during labour, though a satisfying anatomical explanation is forthcoming in the counterpart of this condition, Volkmann's contracture of the flexor muscles of the forearm (Middleton^{s. 9}). The sternomastoid muscle is derived from three different muscular segments. Muscle fibres derived from these segments flow into the neck and unite to form the three portions of the muscle, each portion retaining unaltered its own original arterial supply and consequently its own venous return.

The upper mastoid portion, which is very much shorter than one is generally led to believe, is situated at the mastoid extremity of the muscle, and is supplied by the superior sternomastoid artery derived from the occipital artery. This artery is short and runs a transverse course. The venous return from this portion of the muscle appears to travel through short trunks which penetrate the deep aspect of the muscle and join the large veins beneath. It is difficult to conceive of any mechanism by which these veins could become obstructed, and in fact a careful examination of sternomastoid tumours shows that this small mastoid portion of the muscle appears seldom, if ever, to be affected, and this portion is not the seat of fibrosis in torticollis.

The sternal head of the muscle receives its arterial supply through the middle sternomastoid artery, a branch of the superior thyroid artery. This branch takes origin from its parent trunk a short distance above the upper border of the omohyoid muscle, and the vascular bundle is seen and divided in every case of excision of glands in the neck as it runs backwards and downwards parallel to the upper border of the omohyoid muscle. On reaching the sternal head of the muscle, it does not at once enter it. It passes beneath this head, appearing in the interval between the sternal and clavicular heads. It then courses downwards along the posterior border of the sternal head, giving branches forwards to that head as it goes, but sending no branches to the clavicular head. The venous return from the sternal head appears to follow the artery of supply, and eventually terminates in the venous plexus formed by the junction of the superior thyroid and lingual veins with the common facial vein.

The clavicular head derives its arterial supply from the inferior sternomastoid artery, a small branch coming either direct from the thyrocervical trunk or from one of its branches, usually from the transverse scapular artery. The branch runs upwards upon the deep aspect of the muscle, supplying branches to it. The exact path of the venous return from this head is a little uncertain. Probably much of the blood is returned by a vein accompanying the artery of supply, but Mr. Saunders, of the Anatomical Department of the University of Edinburgh, has observed small venous trunks issuing from the surface of the muscle to join the external jugular vein.

Noré-Josserand and Viannay,¹⁰ investigating the circulation of the sternomastoid muscle in the stillborn foetus, found that these three systems of blood-supply were independent, each supplying its own portion of the muscle and failing to communicate with each other or surrounding arteries. If this is so, it follows, of course, that the venous systems are also independent.

Impressed with the theory of arterial deprivation, they proceeded to show that the arteries to the sternal and clavicular heads can be occluded in the foetus if the head is placed in a position of extreme rotation such as occurs during labour, the tense sternomastoid muscle in this position being capable of compressing the vessels of supply where they pass beneath it. If, under experimental conditions, the middle and inferior sternomastoid arteries can be closed to injection by this means, it is not too much to suppose that the thin-walled veins may be shut off during labour without at the same time interrupting the arterial supply, thus reproducing, temporarily at all events, the condition obtaining in the experimental muscle after venous ligation. It is perhaps hardly necessary to emphasize the fact that such a conclusion must remain at present in the realm of pure theory, but it appears that some such hypothesis offers the only explanation of the origin of the sternomastoid tumour and congenital torticollis which is not seriously at variance with known clinical and experimental facts.

Another point remains to be cleared up in this connection. If sternomastoid tumour results from venous blockage in the positions of rotation assumed by the foetal head during the passage of the pelvis minor, one would expect that such tumours would commonly occur following upon complicated labours, or normal labours which had been unduly prolonged, having therefore an unusually high incidence in primiparous cases. A survey of our series of cases gives us the following information: Out of 64 cases where the place occupied by the child in the family was known, 43 were the first children born to the mother. Of the remaining 21 who were not born of primiparae, 16 were classified as either 'difficult prolonged labours', 'cross-births', or 'breech presentations'. Out of 83 cases in which the history of the labour was known, only 14 were described as normal, 46 were 'difficult' or 'prolonged', 15 were 'breech presentations', and 8 were 'cross-births'.

It is known that obstetrical paralysis of the Erb-Duchenne type almost invariably follows on a difficult delivery succeeding a prolonged and troublesome labour, being especially common after breech presentations. One would expect to find a high proportion of sternomastoid tumours associated with such cases. I therefore went through the histories of 54 cases of Erb's paralysis which had been under observation during the main period (from birth to three months) in which tumours might be found. The series is consecutive except that all cases which were not seen till they were over three months of age are excluded. Out of 54 such cases seen during the last five years, no fewer than 11—that is to say, about 1 case in 5—had exhibited a sternomastoid tumour.

Jepson⁵ has shown that a venous blockage which has been complete for some hours is capable of setting up a process of fibrosis which continues even after the venous obstruction has been relieved. This might quite conceivably occur during labour, but nevertheless one feels that there must be some other factor at work which is capable of rendering such a temporary venous blockage permanent. It seems likely, judging from the appearance of the veins within a few minutes of experimental venous ligation, that thrombosis might well occur in the stagnant blood occupying the greatly engorged venous trunks in the muscle, and in point of fact such thrombosis can be observed

under the microscope in the earlier stages of the process (*Fig. 119*). If the whole venous tree of, for instance, the sternal head of the sternomastoid muscle were blocked for an hour or two during labour, thrombosis occurring in parts of the tree would obviously interfere with the venous return from portions of the muscle permanent, with resulting fibrosis. This would explain the fact that the fibrosis of torticollis is classically patchy in character, with intervening areas of normal muscle. I have carefully examined many sections of muscle from sternomastoid tumours and cases of torticollis in an endeavour to find the remains of these thrombosed veins. One might expect that they would be difficult to find amongst masses of fibrous tissue months or years after the date of thrombosis. And, further, small nerve-trunks on section must be carefully distinguished from them. I have in fact been unable definitely to demon-



FIG. 119.—Microscopical section from experimentally produced tumour of one week's standing. Fibrosis is already advanced, and a thrombus is seen occupying a large vein.

strate such veins either to my own satisfaction or to that of Dr. James Davidson, Pathologist to the Royal Infirmary of Edinburgh, to whom I am indebted for much help on this point.

IV. PATHOLOGICAL CHANGES IN THE SKELETON ASSOCIATED WITH CONGENITAL TORTICOLLIS.

Under this heading we must consider the exostosis which sometimes develops at the attachment of the clavicular head of the sternomastoid muscle, and the facial asymmetry and plagiocephaly which is invariably associated to a greater or less degree with torticollis.

1. **Clavicular Exostosis.**—In most cases of torticollis in which the fibrosis affects the clavicular head of the muscle to any extent the normal markings caused by the muscular attachment to the clavicle are increased, and occasionally a definite exostosis develops at this point. In the more marked cases the exostosis is visible in the X-ray film (*Figs. 120, 121*), and is easily palpable. Such an exostosis is never seen at the sternal attachment, where the fibrosed muscle fibres are separated from the bone by the normal tendon of insertion. It is only met with in the clavicular head, where the scar tissue comes into direct relationship to the bone. It would appear that the new bone formation is dependent upon the minute recurring traumata inflicted upon the subperiosteal layers of the bone through the shortened muscle.

2. **Changes in the Skeleton of the Face and Skull.**—If one examines a patient with congenital torticollis carefully (*Fig. 122*), one will notice in the

majority of cases a more or less well-marked asymmetry of the face and skull. The eyebrow on the side of the shortened muscle tends to slope downwards. The face below the level of the eyes appears to be shorter from above downwards and wider from side to side than on the normal side. In actual fact,



FIG. 120.—H. W., age 7. Torticollis with fibrosis affecting especially the clavicular head of the right sternomastoid. A well-marked exostosis is visible in the radiogram at the clavicular attachment of the muscle. At operation this was removed.



FIG. 121.—Male, age 25, with torticollis. Radiogram of specimen of lower end of sternomastoid removed at operation. A large exostosis is seen to occupy the fibrosed clavicular head. No ossification is present in the sternal head, though the fibrosis was equally intense in both portions of the muscle. (*Mr. J. M. Graham's case.*)

in a well-marked case, the measurement from the external angular process to the angle of the mandible will be found to be shorter on the one side than on the other (Staub¹²).

On turning to the skull one notices that on the affected side the frontal eminence is flattened, while there is a well-marked bulge in the occipital region on that side. Conversely, on the opposite side the frontal eminence is unduly prominent, and the occipital region is, if anything, rather flat. In other words, the vault of the skull has been thrown back on the affected side and forwards on the opposite side, giving rise to a deformity which is, in all essentials, comparable to that seen in the thorax in cases of dorsal scoliosis.

The membranous portions of the skull—that is to say, the bones of the vault of the skull and those of the face—depend for their support upon the cartilaginous base of the skull. Of this base, the essential 'key-stone' is formed by the basi-occiput and the basi-sphenoid. These represent developmentally a modification of the cephalic extremity of the vertebral column



FIG. 122.—Youth, age 18, with torticollis. To illustrate well-marked facial asymmetry.

and form the oldest portion of the base of the skull. On this foundation the anterior and posterior portions of the base are formed by the development of cartilaginous expansions in which the floor of the anterior fossa and the posterior portion of the posterior fossa are laid down. It is clear, then, that any deviation from normal alinement on the part of the basi-occiput and basi-sphenoid will affect the growth of the skull and face as a whole. It is convenient to stress the vertebral origin of the basi-occiput and basi-sphenoid, as it would appear that any curve in the cervical portion of the vertebral column during the early growth period is continued into the base of the skull as a curve which may be associated with a twist corresponding to the well-known vertebral rotation which occurs in cases of scoliosis. The term 'scoliosis capitis', which has been applied to this deformity, appears therefore

to be a strictly accurate one, the deformity of the base being in every way comparable with an ordinary scoliosis, while the asymmetry of the skull and face corresponds to that of the thorax in a case of dorsal scoliosis associated with vertebral rotation. The deformity of the skull varies according to the severity of the cervical curve as long as growth continues, and one therefore finds that the facial asymmetry of torticollis tends to diminish gradually after cure of the torticollis by operation; and indeed, if treatment is undertaken early, all trace of asymmetry may disappear.

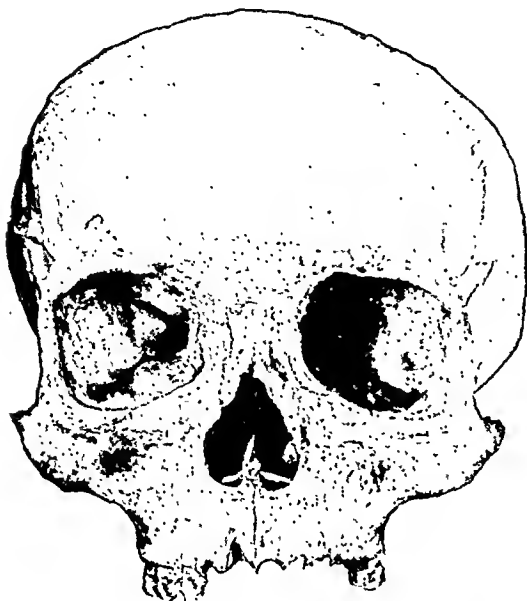


FIG. 123.—Professor Sydney Smith's specimen. *Norma frontalis*. Note the well-marked facial asymmetry.

this light. Children are frequently born with plagiocephaly, more or less severe, which tends to decrease after birth, and in all but the worst cases disappears entirely in the course of a few years. This phenomenon has received many explanations, none of which is satisfactory. In the light of our present knowledge of scoliosis capitis, however, the etiology becomes clear. Such a child has developed in utero with the head in a laterally flexed position, and the skull has consequently grown in an asymmetrical manner. At birth the neck comes straight, and the asymmetry of the skull at once diminishes with further growth.

I have been fortunate in having access to the skull of an adult who suffered from congenital torticollis, now in the Museum of

It is convenient at this point to note that the problem of the so-called 'idiopathic facial asymmetry and plagiocephaly of the newborn' is easily solved when viewed in



FIG. 124.—Professor Sydney Smith's specimen. *Norma occipitalis*. The facial bones have been blacked out in order to show up the hypertrophy of the left (affected) mastoid process, the difference in size between the occipital condyles on the two sides, and the definite rotational twist of the base of the skull.

Forensic Medicine in the University of Edinburgh, which illustrates many of the changes in the skull. I am indebted to Professor Sydney Smith for permission to utilize this specimen. Three photographs are reproduced herewith, and show the angling of the base of the skull in the region of the basi-occiput and the asymmetry of the face very clearly (*Figs. 123-125*). *Fig. 124* shows also that there is a very definite rotational deformity present, the base of the skull being twisted so that the under aspect tends to look towards the side of the affected sternomastoid muscle. In association with this twist there is a marked discrepancy in size between the two occipital condyles.

It is of interest to note that the mastoid process on the affected side is heavy and well formed, while that on the opposite side is unduly small. This is to be expected when one remembers that the mastoid process owes its presence to the pull upon the skull of the sternomastoid muscles when these are developed on the assumption of the erect posture (Darwin² and Schaafhausen¹¹). The mastoid processes are therefore not present in the pronograde vertebrate. The human infant has hardly any mastoid process at birth, and this structure commences to appear when the child learns to hold up his head in the erect position. It is clear, therefore, that in the case of torticollis the shortened muscle will exert a stronger pull upon the mastoid region than the normal one, and the process on the affected side will develop to a greater extent.

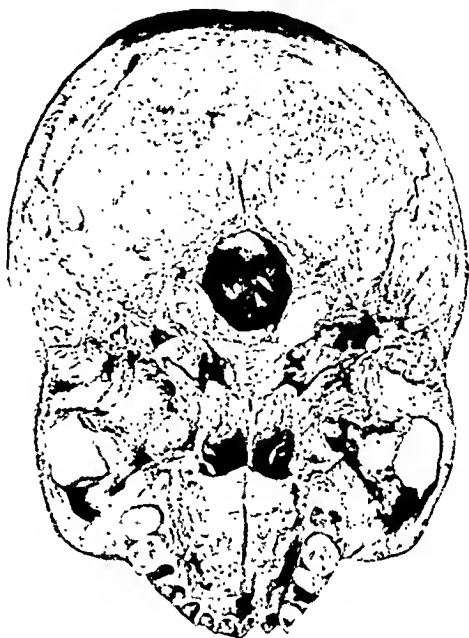


Fig. 125.—Professor Sydney Smith's specimen. Norma basalis. Note the well-marked angle between the anterior and posterior portions of the base of the skull, shown up especially in this view by the gross deviation of the external occipital crest.

CONCLUSIONS.

1. In all cases 'congenital' torticollis results from fibrosis in the sternomastoid muscle consequent upon the development and absorption of the so-called 'sternomastoid tumour' of infancy.

2. Sternomastoid tumour, and therefore torticollis, result from a temporary acute venous obstruction in the muscle, this obstruction taking place during labour.

3. It is probable that such a temporary venous obstruction has been rendered permanent by patchy intravascular clotting in the obstructed venous tree.

4. Attention is drawn to the frequent occurrence of an exostosis at the clavicular attachment of the sternomastoid muscle in cases of fibrosis affecting that head.

5. The facial asymmetry and plagiocephaly associated with torticollis is shown to be dependent upon the prolongation of the cervical curve into the base of the skull, and is therefore properly called a scoliosis capitis.

6. A reasonable explanation is given of the etiology of idiopathic facial asymmetry and plagiocephaly of infants.

I am indebted to Miss Herzfeld and Mr. Norman Dott, of the Royal Edinburgh Hospital for Sick Children, for the generous fashion in which they have referred all cases of sternomastoid tumour and torticollis to me during the course of this investigation, and also to Mr. J. M. Graham for material from adult cases of torticollis.

The expenses of the experimental work were in part defrayed by a grant from the Moray Fund, and I wish to record my thanks to Mr. D. M. Greig, Curator of the Museum of the Royal College of Surgeons of Edinburgh, for much help with regard to illustrations.

REFERENCES.

- ¹ BROOKS, B., *Arch. of Surg.*, 1922, v, 188.
- ² DARWIN, *Descent of Man*, Part I, Chapter 2.
- ³ GALLAVARDIN and SAVY, *Lyon méd.*, 1903, 767.
- ⁴ JEANNEL, *Encyclopédie internationale de Chir.*, v.
- ⁵ JEPSON, P. N., *Ann. of Surg.*, 1928, Dec., 784.
- ⁶ JOACHIMSTAL, *Handbuch der orthopäed. Chir.*, 1904.
- ⁷ KEMPF, *Deut. Zeits. f. Chir.*, 1904, lxxiii, 351.
- ⁸ MIDDLETON, D. STEWART, *Lancet*, 1928, ii, 299.
- ⁹ MIDDLETON, D. STEWART, Jones and Lovatt's *Orthopaedic Surgery*, 2nd ed., 540.
- ¹⁰ NOVÉ-JOSSERAND and VIANNAY, *Rev. d'Orthop.* (2nd ser.), 1906, vii, 399.
- ¹¹ SCHAAFFHAUSEN, *Anthrop. Review*, 1868, Oct., 482.
- ¹² STAUB, *Münch. med. Woch.*, 1921, March 25, 364.
- ¹³ WITZEL, *Deut. Zeits. f. Chir.*, 1883, xviii, 534.

TWO CASES OF HÆMANGIOMA OF THE BLADDER.

By JAS. B. MACALPINE,

SURGEON AT SALFORD ROYAL HOSPITAL, MANCHESTER.

CASE HISTORIES.

Case 1.—A male patient, age 28, complained of symptomless hæmaturia of moderate severity which had recurred on several occasions during a period of three months.

ON EXAMINATION.—The bladder was healthy in every respect save that around the left ureteric orifice were three nævoid patches. They were purplish in colour and consisted of small venous dilatations which projected but little into the bladder. Three groups were seen. The uppermost, about the size of a sixpence, lay above the ureteric orifice, and through the middle of it ran two normal-looking forked blood-vessels of redder colour, apparently arteries. Two smaller groups, each about the size of a pea, lay below the ureteric orifice. All these areas were cauterized with the high-frequency current and the symptoms disappeared. There was no pathological confirmation.

The position of these nævi was similar to that occupied by the submucous hæmatomata of a descending ureteric stone. Symptoms of such a condition had been absent, and the appearance of the lesions did not suggest a submucous hæmatoma, as they were purplish in colour and covered with healthy mucosa, whilst the individual vessels could be clearly identified. They were observed on two occasions several weeks apart and the appearance was identical at each examination, which would not have been the case had they represented a temporary lesion. After cauterization they almost entirely disappeared. This cystoscopic appearance was figured by me in my handbook on cystoscopy.¹

Case 2.—A male, age 14, was admitted to the Pendlebury Children's Hospital suffering from hæmaturia, and was transferred to the urological service at the Royal Hospital in July, 1928. Hæmaturia had occurred at intervals of two to four months since birth, pain on micturition having developed recently.

FAMILY HISTORY.—The patient came of a healthy family, and no urinary disorders were reported. There was no evidence of tuberculosis in the family.

PERSONAL HISTORY.—He had suffered from diphtheria, scarlet fever, measles, and chicken-pox. He had a congenital dislocation of the right hip, with a false joint; over this an abscess developed and was incised at the Children's Hospital. Movements of the hip were good up to a right angle, and the hip could be slightly abducted; there was two inches of shortening.

ON EXAMINATION.—The boy was badly nourished, anæmic, and thin. The tongue was slightly coated and the teeth were carious; heart and lungs were normal. Examination of the abdomen showed no abnormality, and the kidneys could not be palpated. There was a severe degree of what appeared to be internal hæmorrhoids. In the left scrotum a venous mass was palpated which felt like an ordinary varicocele. The left testicle was soft and atrophic. The right testicle was about the size of a pea, but was in the normal position: it had apparently no testicular sensibility. In the epididymis a cyst the size of a bean was felt. On the dorsum and lateral aspects of the glans penis the submucous vessels were diffusely dilated, suggesting a cavernous angioma. On the dorsum of the penis itself was a firm plaque of slightly purplish colour the size of a small almond which occupied the subcutaneous tissue. On the outer side of the right thigh were two large irregular

areas, each about three to four inches in diameter, lying deeply underneath the skin. They were diagnosed as cavernous angiomas. They were soft, and collapsed on pressure, leaving hollows in the site they had previously occupied. A bandage was worn over them, as they ached when the patient occupied the upright position for long. A scar over the posterior aspect of the right hip indicated the site of incision of the above-mentioned abscess.

Urine.—The urine contained albumin in fair quantity, and pus, together with occasional blood. Its reaction was acid, and the specific gravity varied between 1010 and 1020. Microscopical examination showed numerous pus-cells and a fairly large number of red blood-corpuscles. No casts and no tubercle bacilli could be found; cultural methods revealed a growth of the *Staphylococcus aureus*.



FIG. 126.—Appearance of hemangioma of the bladder after removal.

Radiography.—A skiagram of the urinary tract showed several shadows on the right side at the level of the second and third lumbar vertebrae which were regarded as being outside the urinary tract. The radiographical examination was otherwise negative.

The Blood Urea was 40 mgrm. per 100 c.c. of blood.

The urea concentration on July 7 was :—

Before administration of urea	1.75	per cent
1st specimen	1.75	" "
2nd specimen	3.1	" "
3rd specimen	3.4	" "

The temperature and pulse were normal.

Cystoscopy.—On cystoscopy the bladder held about 6 oz. and the ureteric orifices were similar and normal. A large tumour was observed immediately behind the air-bubble, occupying the middle line and extending down towards the 'bas fond'. It was for the most part reddish in colour, and had numerous obviously vascular areas on its surface, between which were patches of sandy yellow colour which projected into the bladder cavity and had a convex intravesical surface. Projecting from it were a large number of tags of *adenomatous mucosa* not unlike the villi of a papilloma. No central blood-vessel was observed in these, but a few of them appeared to have extravasated blood in their interior. Many of them were club-shaped and stunted. No cystoscopic drawing was obtained, but the characters of the tumour can be judged from the drawing of the fresh specimen (*Fig. 126*).

OPERATION (July 6).—The bladder was filled with 6 oz. of solution, and a mid-line suprapubic incision extending from the pubis to the umbilicus was employed. On exposure of the bladder a tumour half the size of a tennis ball was discovered on its upper and posterior surface, partly covered by peritoneum and partly in front of the peritoneal reflection. The tumour formed a projection half an inch in depth beyond the external surface of the bladder. The urachus was thickened and entered its posterior part about the mid-point. It was cut close to the bladder.

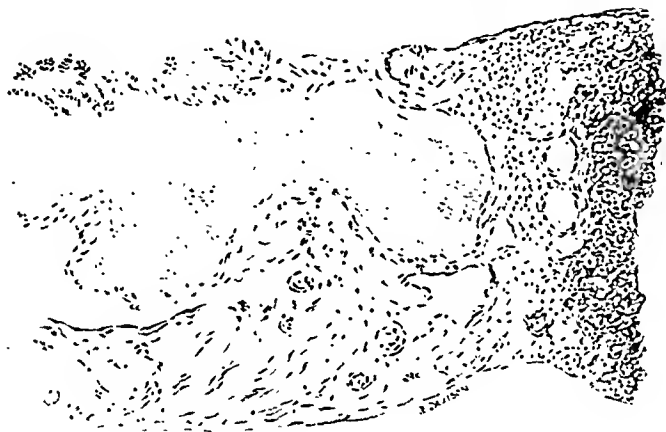


FIG. 127.—Microscopical appearance of hæmangioma of the bladder.

The bladder was mobilized on its lateral aspects, and the peritoneum was then opened. Investigation of the intraperitoneal structures showed a *nævroid* condition of the transverse colon from its middle point onwards to the descending colon and to the rectum. A similar condition was felt in the retroperitoneal structures internal to the colon. The *nævroid* tissue in the rectum was evidently continuous with the hæmorrhoidal tissue previously remarked. The bladder, having been freely mobilized, was opened, and the tumour was found to project into the cavity for one to one and a half inches. Its margins were moderately well defined, there being no evident cavernous tissue beyond them. It was excised together with a margin of healthy bladder wall. Bleeding was not unduly troublesome nor was hæmostasis difficult. The bladder was re-formed, an in-lying catheter inserted, and the wound was closed in layers, a tube being left in the suprapubic space.

PROGRESS.—Convalescence was uneventful except for slight infection of the abdominal wound round the area previously occupied by the suprapubic tube. The patient was discharged on Aug. 6, 1928, the wound being healed. The bladder capacity was approximately 4 oz. and the urine clear.

Since then there has been no hæmaturia. Micturition occurs at the normal intervals, and the patient is comfortable and well. Fresh cystoscopy has been refused. There is a tendency for the angiomatous tissue on the glans penis to become more pronounced.

The accompanying coloured drawing (*Fig. 126*) shows the specimen after removal. The irregularity of the surface is evident, and numerous vascular protrusions are well shown. The surface was seen at cystoscopy to be covered with villiform processes, and these can be detected in the drawing. In actual fact they fell flat when the specimen was displayed for the artist, and had to be floated up in fluid in order to allow her to identify them. They were more numerous than would appear from the drawing. The fact that the tumour is sharply circumscribed is also evident.

I am indebted to Professor Shaw Dunn for cutting sections and making the following report:—

Tumour of the Bladder Wall.—"Histological examination of a large block taken to include the margin of the growth shows that it is composed of large cavernous vascular channels. These contain blood and are lined by a single layer of endothelium: their walls are composed of rather hyaline fibrous tissue and show no intrinsic muscle. Muscle bands of the bladder wall pass among the vascular channels in all directions, showing that the tumour is closely incorporated with this tissue. The structure is typical of a cavernous hæmangioma." (*Fig. 127.*)

REVIEW OF THE LITERATURE.

The rarity of this condition is attested by all writers. I have collected from the literature 19 cases, of which the first (Broca) is of very doubtful authenticity.

Broca² is generally referred to as having discovered the first angioma, but his case is of doubtful authenticity, and is regarded as a papilloma by Pousson. The tumour was the size of a hazel nut, and consisted of a network of dilated capillaries. It was discovered at autopsy on an old woman.

Langhans³ observed a cavernous tumour in the bladder of a 19-year-old youth who had periodically suffered from hæmaturia since the age of 10. This specimen also was discovered at autopsy, the latest bleeding having been so severe as to end fatally, and having apparently been excited by alcoholic excess. Several cavernous tumours, of which the largest lay above the left ureteric orifice, were found. At other parts of the bladder the subserous veins were dilated and tortuous. More than one of these showed rupture, with plugging of the orifice by a clot. Accurate histological description was rendered difficult owing to faulty preservation of the specimen.

Albarran⁴ reported a case of a 64-year-old man who suffered from profuse symptomless bleeding and died at operation. At the post-mortem a retro-trigonal tumour the size of a pea was found in an otherwise healthy bladder. Microscopically it proved to be a cavernous angioma, in which sinuous capillaries with ampullary dilatations were separated from each other by thin connective-tissue septa.

Arbuthnot Lane⁵ reported a child 3½ years of age who for two years had suffered from hæmaturia of varying severity. The skin over the gluteal regions showed several small cutaneous nævi. Suprapubic cystotomy revealed vascular masses which protruded into the wound. Almost the whole of the viscus was affected. On account of this widespread involvement no treatment was undertaken, the bladder being closed. Cessation of the bleeding and improvement in health followed the operation.

Berliner⁶ reports an 11-year-old schoolgirl who suffered from her fourth year onwards with intermittent hæmaturia which had recently increased considerably in intensity and caused a severe degree of anæmia. Pain and difficulty in micturition had made their appearance latterly. Cystoscopy showed numerous large circumscribed purple swellings and rounded elevations, some of them the size of one's little finger. Only small areas at the apex of the bladder were unaffected. Operation—cystotomy and cauterization—was performed, with cessation of the bleeding to date of publication. Other lesions: (1) On the right labium majus a telangiectatic angioma the size of a walnut (cauterized); (2) The face was strikingly asymmetrical, the left side appearing to be swollen owing to hypertrophy of the parotid and lower jaw. Six months after the operation investigation showed that a stone had formed on a silk suture and necessitated crushing.

Bryan⁷ reported a man, age 35, who complained of hæmaturia. Cystoscopically a broad sessile tumour was seen to be present at the back part of the bladder. It was as big as a thumb-nail and projected into the bladder. It was removed through a suprapubic incision by blunt dissection and the base was cauterized. Microscopically a cavernous angioma was discovered and is illustrated.

Thunum⁸ reports the history of a 45-year-old woman who for years had experienced pain on micturition. Cystoscopy showed a small tumour, situated 2 cm. behind the ligamentum interuretericum, which consisted of three parts, two of which exhibited an intense blue coloration. The symptoms abated and no operation was undertaken.

Bachrach⁹ discovered cystoscopically what was apparently a cavernous angioma in the bladder of a 13-year-old girl. The patient had had vaginal hæmorrhage at the age of 4 and again at the age of 9. For the last one and a half years there had been irregular bleeding from the vagina which had been regarded as menstrual, but which on one occasion had been unduly severe. Hæmaturia showed itself when the girl was 13 years old, together with pain in the thigh and frequency of micturition. In the upper left quadrant of the bladder a tumour the size of a cherry-stone was seen through the cystoscope to project into the bladder cavity. It was in part transparent and in part opaque (? thrombosis). It was vascular in character. Treatment: general. No operative or pathological confirmation was obtained. On the outer side of the thigh extensive nævi were observed, and these are illustrated. They extended from the trochanter to below the knee. Presumably the uterine hæmorrhage arose from nævoid lesions in the womb.

Blum,¹⁰ in the series of cases which he reported in his monograph on bladder diverticula, describes that of a man, age 64, who for four years had suffered from hæmaturia of variable intensity which was becoming progressively worse. Recently he had developed a severe anæmia. At operation the diverticulum was excised, and an angioma the size of a lentil was found projecting from the anterior wall. This had been invisible at cystoscopy owing to its position within the diverticulum. Blum remarks that this could only have been discovered cystoscopically had the cystoscope been introduced within the diverticulum.

Launay, Aclard, and Carrière¹¹ report an angioma discovered accidentally

during an operation for salpingitis and appendicitis. On opening the peritoneum a firm lobulated tumour the size of an orange was observed embedded in the musculature of the bladder. Operation: partial cystectomy, salpingectomy, appendicectomy. The vesical surface of the tumour was smooth, and covered with healthy mucosa. A section running from the vesical to the peritoneal surface showed the latter area to be formed of fibrous tissue, and from it septa diverged towards the mucous surface, dividing the tumour into lacunæ of various sizes, of which the least was about 5 mm. in diameter. Some of these were filled with blood-clot, others with a gelatinous substance of greenish colour. All the cavities were lined with a single layer of flattened endothelium. (Illustrated.)

Rafin¹² reports the cystoscopic appearances in the bladder of a girl of 17 who suffered from severe intermittent hæmaturia. The trigone and ureteric orifices were normal. Part of the remainder of the bladder was covered with red granular elevations separated from each other by small intervals of pale colour. At other portions of the bladder "large bluish vessels forming veritable venous sinuses" were observed. Some appeared to be on the point of ulcerating. He regards it as a "diffuse venous nævus". There was no operation and no pathological confirmation.

Sotti¹³ describes in great detail a specimen removed post mortem from the body of a woman, age 36, who died of bronchopneumonia. There was no record of any bladder complaint. The angioma was at first sight judged to be a papilloma. It was about 13 mm. in diameter, and occupied the base of the bladder, abutting on the neck, and encroaching for a short distance posteriorly on the urethra. Its surface was rounded, and covered with smooth, glistening, healthy mucosa the vessels of which were normal in type and did not resemble those of the growth. The tumour was closely incorporated with the vesical musculature. Its microscopic appearances are recorded at great length, and are those of a cavernous angioma with considerable development of elastic tissue.

Hübner¹⁴ describes a female patient 11 years of age who had suffered from the age of 6 with hæmaturia. She was pale, thin, and anæmic. Cystoscopic investigation showed a cavernous angioma lying behind the air-bubble, consisting of tensely filled venous dilatations some of which were isolated whilst others were confluent, and the venous dilatations appeared like a row of pearls. The tumour extended down within 3 cm. of the right ureteric orifice. Treatment consisted of partial cystectomy; that part of the growth which lay close to the right ureteric orifice was not removed. There was strikingly little bleeding from the bladder incision. The pathological examination showed a cavernous angioma with much elastic tissue in the septa. Some weeks later this patient developed a fresh hæmaturia, and it was found that those portions which could not be removed had developed fresh angiomatous tissue. They were treated by thermo-coagulation, and after three sessions the bleeding ceased.

Faerber¹⁵ has reported what appears to be the same case as the above.

Kidd and Turnbull¹⁶ report a case which they call an angiomoma, the patient being a male, age 29 years. A smooth pedunculated tumour the size of a date and looking like a raspberry was attached to the anterior margin

of the internal meatus by a narrow stalk. Its apex was ulcerated. Histologically the tumour showed itself as "an excrecence of muscularis, mucosa, and epithelium supplied with abnormally large and numerous vessels" . . . "the muscle bundles are not connected with the formation of the vessels as is the case in some angiomata; the vessels are perfectly formed, and are absolutely distinct structures". This tumour does not appear to be quite comparable with the ones we have described.

Scholl,¹⁷ in an article on histology and mortality in cases of tumour of the bladder, has reported three cases under the heading of angioma, two of which do not appear to be relevant to the present paper, the first being an "inoperable sessile growth covered with papillomatous excrecences", and the second a "highly vascular myxoma". A third patient was a girl of 7 who had had persistent slight hæmaturia for six years. Recently a small amount of blood had been passed from the rectum. Cystoscopy revealed an ulcerating bleeding tumour in the base of the bladder. Soon after, and before operation could be performed, the patient had a severe hæmorrhage from the rectum and died. Necropsy revealed an extensive cavernous angioma growing from the base of the bladder and invading the rectal mucosa. No microscopical description is given, nor any figure.

Thomas¹⁸ reported a small angioma of the bladder which was cured by fulguration. There was no pathological confirmation.

Caulk¹⁹ reports two cases in his practice. The first, a tumour having "the appearance of an ordinary nævus", occurred in a man, age 35. It was completely destroyed by the high-frequency current. No microscopic confirmation is given.

The second case was a woman, age 60, who suffered from hæmaturia. On vaginal examination there was a very hard indurated area on the vault of the vagina corresponding to the base of the bladder in the region of the trigone. This subsequently proved to be a large calcified lymph gland, which was located near the juxtavesical ureter and which was dissected out. Cystoscopically a peculiar condition of the left lateral wall near the ureteral orifice was observed. It had a mottled appearance, consisting of areas of blue and almost black spots intermingled with lighter cystic-looking masses. At the outer edge of the mass there was a typical telangiectatic spider-web dilatation of the blood-vessels. At operation it was possible to dissect out the whole tumour, which was found to encircle the lower portion of the ureter and was adherent to the adventitia of that tube. It consisted of irregular conglomerated masses of vascular dilatations which on pathological examination proved to be a characteristic hæmangioma of the cavernous type. The author regards this case as an angioma of the ureter, and it is apparently unique.

Katz²⁰ relates the case of a man, age 40, who complained of intermittent hæmaturia for six months. It appeared only at the end of micturition. The patient was an unfavourable subject for operation on account of cardiac and respiratory lesions, a moderate degree of anæmia, and bilateral renal stones. Cystoscopy revealed a bleeding point in the vicinity of the internal meatus, no blood coming from the ureters. Severe hæmorrhage compelled operative treatment, which took the form of cystotomy and

Paquelin cautery to the bleeding area near the internal meatus. Copious bleeding recurred within a few hours, and the patient died. Microscopical investigation showed "variously sized vascular lacunæ which were lined by endothelium . . . some of them penetrated the upper muscular layer". (Photomicrograph.)

Maedonald²¹ showed a specimen of "nævus of the bladder" on Nov. 28, 1929, at the Royal Society of Medicine, which is included in their *Proceedings*. The tumour was excised in June, 1929, from the bladder of a male patient, age about 33. The only symptom was slight terminal hæmaturia, beginning about a month previously. Cystoscopy showed a tumour, apparently a nævus, growing from the left upper quadrant of the bladder. Microscopically the tumour is a cavernous angioma.

COMMENTARY.

There are, as we have seen, 20 cases on record in addition to the two reported here, though Broea's case was probably not an angioma. Of these 20 cases, only 8 have been confirmed by histological investigation. A surprisingly large number of cases—9—cannot claim histological confirmation, either because they were reported prior to the days of systematic submission of material to the pathologist, or because there was no open operation. Small nævi suitable for per-urethral diathermy will probably continue to appear in the literature without histological investigation. To this category belongs my first case, and also Thomas's and one of Caulk's cases. Moreover the same lack of confirmation will exist whenever treatment consists of cauterization through a suprapubic incision, as in Lane's and Katz's cases, the fear of hæmorrhage prohibiting the excision of portions for investigation. The diagnosis in all such cases must of course be accepted with reserve.

In the earlier literature a curious tendency is observed to confound vesical varices with true angiomata. It is necessary on this account to exclude various reports in which the lesion was obviously a varix. The *Encyclopédie française d'Urologie* calls attention to this: "Certain authors regard varices as tumours which should be ranged alongside angiomata. This opinion cannot be seriously defended. Varices are dilatations and vascular hypertrophies without new formation of vessels." Casper, however, reports an evident varix under the heading of an angioma, as do numerous other writers, and Sotti considers that the differentiation between the two is not always easy even after histological investigation. The differentiation, however, should be possible cystoscopically, and is important, because the clinical course of the two diseases is different. The bleeding occurs once or twice and tends not to be repeated when coming from a varix; whilst, as is evident from a study of our collected cases, the hæmorrhage from angiomata invariably recurs, is frequently profuse, and in four instances (Albarran, Langhans, Scholl, and Katz) led to death from exsanguination. Further, it may be considered justifiable to adopt expectant treatment in the case of varices, while more active measures are obviously required for angiomata.

The existence of nævoid tissue elsewhere was a striking feature in my second case. Extensive involvement of the colon, rectum and anus, thigh, scrotum, and penis was observed. In none of the other reported cases has there been such widespread hæmangiomatous tissue, though in four instances other nævi were reported. In Berliner's case a telangiectatic angioma the size of a walnut was present in the right labium. Lane's patient had venous lesions on the skin of the buttock, and Scholl's died of a hæmorrhage from the rectum, whilst Bachrach's patient had extensive nævi on the thigh and presumably a uterine nævus. Evidence of true cavernous tissue in other parts of the body must be sought for. It should be regarded as confirming the diagnosis of a cavernous tumour in the bladder, and helps in the differentiation of vesical angiomata from vesical varices. The simultaneous occurrence of angiomata in widely separated organs or surfaces is well known. Kramer,²² in an article on angiomata, recorded 147 cases in which 28 per cent showed multiplicity of the lesions, and Ullmann²³ recognizes a condition of universal angiomatosis. Varices moreover occurring in the bladder are very prone to be associated with similar lesions—generally extensive—on the thighs, labia, etc.

My second patient also exhibited congenital deformities in the dislocation of the hip and the non-development of the right testis. It might have been expected that abnormalities would occur in other reports, but the only one which I have noted is that by Berliner, in which facial asymmetry was remarked.

Symptomatology.—A study of the recorded cases shows that hæmaturia is the outstanding symptom, though in many application for treatment was precipitated by the onset of vesical pain and frequency, which usually corresponded to the occurrence of infection. Once bleeding is established it tends to be continuous, or to recur at intervals and become increasingly severe. In several cases it was so profuse as to lead to a fatal issue, and in many cases to a severe grade of anæmia. The occurrence of nævi in other situations and of congenital deformities has already been discussed.

Treatment.—The treatment of these cases will vary according to their size and site. Per-urethral diathermy is suitable for small and obviously superficial tumours. It should be remembered, however, that the main bulk of the tumour, especially of the cavernous tumours, occupies the muscular planes (*see* Hübner, Sotti, Launay, and my own report), and that cauterization will almost certainly open up deep vessels which cannot be sealed by the electrode. Hæmorrhage following open cauterization recurred in Katz's case and terminated fatally.

Excision of the mass, as done by Launay, Hübner, Kidd, and myself, is a superior method when the circumstances permit. Apparently no anxiety need be felt regarding difficulty in hæmostasis when partial cystectomy is undertaken. In my own case the cut bladder wall appeared to be no more vascular than it does in ordinary circumstances, and Hübner and Launay observed and emphasized the same freedom from excessive hæmorrhage. An attempt should be made to remove the whole of the tumour and with it a fair margin of healthy tissue. In Hübner's case this was impossible, and a footnote added after the paper was in print showed that a recurrence had taken

place with severe bleeding, thermo-coagulation on three occasions being required to bring about a cessation of the symptoms.

REFERENCES.

- ¹ MACALPINE, J. B., *Cystoscopy*, Plate VI F. Bristol: John Wright & Sons Ltd.
- ² BROCA, *Traité des Tumeurs*, II.
- ³ LANGHANS, "Kasuistische Beiträge zur Lehre von den Gefäßgeschwulsten", *Virchow's Arch.*, 1879, lxxv, 391.
- ⁴ ALBARRAN, *Les Tumeurs de la Vessie*, 1892. Paris.
- ⁵ LANE, *Lancet*, 1895, 1252.
- ⁶ BERLINER, "Die Teleangiektasien der Blase", *Deut. Zeits. f. Chir.*, 1902, lxiv, Parts 5 and 6.
- ⁷ BRYAN, *Southern Surg. and Gyn. Trans.*, 1909, xxii, 518.
- ⁸ THUMM, "Zur Kenntnis der Hämangiome der Harnblase", *Verhandl. de deut. Gesellsch. f. Urol.*, 1909, 368: also *Zeits. f. Urol.*, 1909, 368.
- ⁹ BACHRACH, "Ueber Teleangiektasien der Harnblase", *Fol. urol.*, 1910.
- ¹⁰ BLUM, *Chirurg. Pathologie und Therapie der Harnblasendivertikel*, 1909. Leipzig.
- ¹¹ LAUNAY, ACHARD, H. P., and CAMILLE, C., "Une Observation d'Angiome de la Paroi vesicale", *Jour. d'Urol.*, 1920, ix, 385.
- ¹² RAFIN, "Angiome diffus de la Vessie," *Ibid.*, x, 315.
- ¹³ SOTTI, "Contributo allo Studio ed alla Conoscenza dell'Emangioma cavernosa della Vesica urinaria", *Pathologica*, 1921, xiii, 135.
- ¹⁴ HÜBNER, "Das kavernöse Angiom der Blase", *Arch. f. klin. Chir.*, 1922, cxx, 575; "Hemangioma of Bladder", *Zeits. f. Urol.*, 1923, xvii, 29.
- ¹⁵ FAERBER, "Ein Fall von Haemangiom der Harnblase bei einem elfjährigen Mädchen", *Forts. d. Med.*, 1922, xl, 358.
- ¹⁶ KIDD, F., and TURNBULL, H. M., "Angiomyoma of the Urinary Bladder", *Surg. Gynecol. and Obst.*, 1923, xxxvi, 467.
- ¹⁷ SCHOLL, "Histology and Mortality in Cases of Tumor of the Bladder", *Ibid.*, 1922, xxxiv, 189.
- ¹⁸ THOMAS, quoted by CAULK.
- ¹⁹ CAULK, "Hemangiomata of Bladder and Ureter", *Surg. Gynecol. and Obst.*, 1925, xli, 49.
- ²⁰ KATZ, "Cavernous Hemangioma of the Bladder", *Jour. of Urol.*, 1926, xv, 201.
- ²¹ MACDONALD, S., "Nævus of the Bladder", *Proc. Roy. Soc. Med.*, 1929.
- ²² KRAMER, BRUNHILDE, "Ueber Hämangiome", *Jahrb. f. Kinderheilk.*, 1904. Quoted by Hübner.
- ²³ ULLMANN, "Ueber einen Fall von multipler eruptiver Angiombildung im Gesicht", *Arch. f. Dermatol. u. Syph.*, 1896.

THE ANATOMICAL RESULT OF PERI-ARTERIAL SYMPATHECTOMY.

By D. M. BLAIR,

PROFESSOR OF ANATOMY, KING'S COLLEGE, UNIVERSITY OF LONDON:

D. DUFF,

SURGEON TO GLASGOW ROYAL INFIRMARY:

AND J. A. BINGHAM,

SURGEON TO OUT-PATIENTS' DEPARTMENT, GLASGOW ROYAL INFIRMARY.

OF recent years operative procedures upon the vasomotor nerves have been increasingly made use of in endeavouring to alleviate or cure a variety of clinical conditions. The principle underlying these operations is the physiological observation that division of the vasomotor nerves is followed by a peripheral vasodilatation. The usual method is to perform peri-arterial sympathectomy proximally in a limb with a view to causing increased vascularity distally. The results have been notably varied in all conditions for which the treatment has been tried, and the uncertainty of result may be correlated with our present lack of precise knowledge of the origin, distribution, and function of the vasomotor nerves. Experimental study of the vasomotor nerves in animals can yield useful information, but it has been noted (Woollard,¹ 1926) that the arrangement of the nerves varies in different species. Direct observation on human material is also called for. In order to supplement the information gained by ordinary dissecting methods (such as the work of Potts,² 1914, and Kramer,³ 1914), no opportunity should be lost of making what amount to experimental observations, where possible, in material arising in the clinical use of sympathectomy operations. When it is subsequently necessary to resort to amputation, investigations of the anatomical result of the previous operation upon the limb may add to our knowledge of this difficult problem. It has been thought desirable to record even the limited observations made in one such case, in the hope of stimulating further similar investigations where opportunity offers.

HISTORY.—Mrs. P., age 57, was admitted to Mr. Duff's Surgical Unit at the Glasgow Royal Infirmary on Oct. 2, 1926. She had suffered from diabetes mellitus for twelve years and had been receiving insulin for one and a half years. Nine weeks before admission gangrene began in the right foot, accompanied by much pain.

ON ADMISSION.—The patient was in poor general condition and suffering severe pain. There was an area of moist gangrene the size of her hand on the dorsum and outer side of the foot, overlapping the external malleolus. The foot elsewhere was cold and blue, the vessels of the limb were sclerosed, and popliteal pulsation was just perceptible. The urine contained abundant sugar and albumin.

During the next two months the glycosuria was controlled by insulin, and local surgical measures were applied to the foot. Sloughs were gradually removed until a sluggish ulcer remained, making no progress. Morphine was occasionally required for the relief of pain. A trial of peri-arterial sympathectomy was decided upon, and the following extracts from the case record indicate the effect on the leg:—

Dec. 13.—Mr. Duff exposed the right femoral artery in Hunter's canal and injected absolute alcohol round the adventitious coat of the vessel. No immediate change in the foot was noted.

Dec. 14.—Foot appears decidedly warmer and colour is better. Pain is less. This progress was maintained during following days.

Dec. 20.—Granulations of ulcer are much healthier. Foot is warm and colour is good. Not much pain.

Dec. 28.—Foot continues to improve.

Jan. 6, 1927.—Improvement has ceased. Pain has returned. Foot feels colder.

Jan. 13.—Foot has relapsed to condition as before operation. Pain is severe. Amputation has been advised.

Jan. 17.—Mr. Duff amputated through thigh just above site of sympathectomy.

The limb was obtained immediately on amputation, and histological examination of the nerves on the main arteries was undertaken (D. M. B.).

The whole of the femoral artery was removed with its adventitia. It was estimated that the artery had been divided about one inch proximal to the site of previous alcohol injections, but owing to the necessary clearing of the adventitia to permit of ligating the vessel before dividing it, only a very short portion of artery with undisturbed adventitia was obtained above the injection site. The femoral trunk was split into longitudinal halves, one immersed in dilute methylene-blue solution for supravital staining, the other prepared by Ranson's method and divided into short lengths which were serially sectioned. An attempt to stain the remaining arteries of the limb with methylene blue by injection from the upper end of the popliteal artery was unsuccessful—probably owing to impermeability of the sclerosed walls. A block dissection of the popliteal artery and the upper ends of the anterior and posterior tibial arteries was, however, successfully stained by immersion. Pieces of both tibial arteries were prepared by Ranson's method. In addition, sections of all the arteries mentioned and of the peroneal artery were also stained with hæmalum and eosin in the ordinary way. By using both the supravital methylene-blue method and Ranson's method of nerve staining it was possible to examine the tissues both by dissection under the binocular microscope and by serial sections, and the results obtained by one method could be checked by the other.

Throughout all the vessels examined degenerative changes were widespread. There was much patchy calcification and apparent thinning of the tunica media, with asymmetrical thickening of the tunica intima. The peroneal artery was least affected, but in the lower parts of both tibials the intima was thickened all round almost to complete obliteration of the lumen.

With regard to the condition of the nerves accompanying the arteries, the extensive calcareous change in the vessel walls prevented study of the finer intramural arrangements of the vascular nerves, but the following points were established.

The longitudinal half of the femoral artery stained by methylene blue and dissected under the binocular microscope proved to contain the greater number of the larger vascular nerve-trunks (the asymmetrical distribution of these trunks was confirmed in sections taken from a normal human femoral artery). Four to five nerve-bundles could be made out, anastomosing with one another to form elongated meshes along the vessel. They ran in the inner face of the separated 'sheath' of the artery. (When an artery is 'cleaned'

its 'sheath' is separated by splitting the adventitia between the narrow inner zone of compact fibrous tissue and the wider outer zone of much looser texture.) Immediately above the site of injection the nerve-fibres were rather faintly stained, a few showing normally stained axons, but from the injection site downwards there was a complete absence of all axon-staining in the nerve-bundles. In sharp contrast to this, towards the lower end of the femoral artery appeared a small nerve-bundle with well-stained axons, running in to the adventitia from without, at first accompanied by a small artery. On penetrating deeper into the adventitia the fibres of this bundle appeared to distribute themselves distally within two of the main degenerated nerve-trunks. They were then difficult to follow, but were traceable to the lower end of the specimen.

The other longitudinal half of the femoral artery, stained by Ranson's method, showed similar features. Above the injection site three larger nerve-trunks were seen, all having a matted appearance, with loss of distinctness in the nerve-fibres and only a very few axons staining. In marked contrast with these, a fragment of the long saphenous nerve included in the block showed good normal staining. An occasional minute nerve-bundle immediately outside the muscular coat seemed normal. Below this level all the perivascular nerves identifiable in the sections showed degeneration, except two incoming twigs. The upper was accompanied by a small artery; the lower approached the femoral artery immediately above the origin of a large muscular branch (? the anastomotica magna). Several fasciculi of the incoming nerve surrounded this arterial branch, which then appeared to carry off with it a comparatively normal nerve-supply. Only a small portion of the new nerve remained with the trunk of the femoral, its fibres ultimately becoming lost in the adventitia.

The popliteal artery (haemalum and eosin sections) showed several larger nerves running into the outer part of the adventitia, all of normal appearance, with a preponderance of small medullated fibres, and all associated with small arteries. Deep in the adventitia were several smaller nerves, all showing degenerative appearances—the fibres seeming denser and more deeply stained than normal, and having blurred, irregular outlines. The lower part of the popliteal and the commencement of the anterior and posterior tibial arteries (methylene-blue dissected preparation) showed an abundant supply of nerves with well-stained axons, in marked contrast to the appearances seen in the femoral artery.

The posterior tibial artery had both normal and degenerated nerve-fibres in its wall. One large nerve running between the artery and its venæ comites contained well-stained axons except in one small degenerated portion. The normal fibres were chiefly of small medullated type, with a few large medullated fibres and some non-medullated fibres. The posterior tibial nerve, included in one block, showed, by contrast, a predominance of large medullated fibres. Deep in the adventitia were small nerve-bundles, some of normal appearance, others degenerated. All the nerves in this region (including the posterior tibial) showed thickening of the endoneurium, and in the adventitia of the artery were regions of small round-celled infiltration.

The peroneal artery showed a few small nerves in the adventitia, all of

normal appearance, without endoneurial thickening, and again with a preponderance of fine medullated fibres. No degenerated nerve-fibres were seen. The condition of the nerves upon the anterior tibial artery was not accurately ascertained. Some small nerves running deeply in the adventitia showed chiefly the fine medullated type of fibre.

These observations show that, although all the nerves running with the femoral artery at the seat of the original operation appeared to have been destroyed, fresh accessions of nerve-fibres arrived distally. A few small twigs joined the femoral artery in Hunter's canal, but the greatest influx of additional nerves occurred in the popliteal space. In all these incoming nerves whose composition could be definitely noted there was a predominance of fine medullated fibres (1.5 to 3 μ in diameter), with a few large medullated fibres (4 to 8 μ) and a distinct contribution of non-medullated fibres.

A special dissection of a normal human leg was undertaken (J. A. B.) to see how far the number and source of the incoming nerves to the blood-vessels could be recognized by ordinary macroscopic methods. The main vessels from the commencement of the common femoral artery to the level of the ankle-joint were dissected, together with the main nerves of the limb; great care was taken not to disturb the relations of these nerves to the arteries until all possible twigs from the former to the latter had been identified. The bones were disarticulated and shelled out from the muscles where necessary, to permit this being done. Every suspected nerve to an artery was carefully dissected out with the aid of binocular forehead magnifiers, and its origin from a nerve-trunk and true termination in the wall of an artery fully established. No nerves were found running to the sheath of the common femoral or the superficial femoral artery in Scarpa's triangle. In Hunter's canal two distinct twigs from the saphenous nerve joined the artery, one in the upper part of the canal, the other an inch and a half lower down. No supply from any other adjacent nerve was found. Immediately above the bifurcation of the great sciatic nerve there arose from the front of the nerve a long slender branch which appeared to lose some of its filaments in the connective tissue of the popliteal fossa; but the majority of its filaments were distributed to the sheath of the popliteal artery just above its bifurcation. This nerve could be traced into the internal popliteal division of the sciatic nerve. From the deep surface of the internal popliteal nerve there arose a definite leash of small nerves, of which some were distributed to the anterior tibial artery, but the majority went to the posterior tibial artery. Four small twigs from the posterior tibial nerve ran to the sheath of the posterior tibial artery in the middle two-fourths of the calf of the leg. No further twigs were found going to the anterior tibial or peroneal arteries.

The findings in the dissected and 'experimental' limbs correspond fairly closely, and are in general agreement with the results obtained by Potts (1914) in dissections of nerves to the arteries of the leg. Neither we nor Potts observed any vascular twig coming from the obturator nerve in the thigh. It seems a pity if the only vascular nerve in the leg which is mentioned in the ordinary text-books of anatomy has to be dropped.

This double source of vascular nerves—from the aortic plexus running peri-arterially into the limb, and incoming in the limb from the ordinary

RESULT OF PERI-ARTERIAL SYMPATHECTOMY 219

nerve-trunks—was also recognized by Woollard (1926) in animals. In the cat, where he put the matter to experimental proof, the transition between the two types was found to occur about the level of the bifurcation of the common femoral artery. He thought the change might occur lower down in dogs. He states that the supplementary nerves were chiefly medullated, but that the peri-arterial trunks were almost entirely composed of non-medullated fibres. The nerve-trunks found with the upper part of the femoral artery in the case here examined were certainly not wholly or in great part non-medullated, but our investigation does not show how far these nerves may have come from the aortic plexus, or from, say, the femoral nerve above Scarpa's triangle. It seems to us rather that the nerves running peri-arterially into the leg, and those reaching the vessels distally from the nerves of the limb, are similar in structure. There is no sharp transition line indicating a proximal supply area belonging to the first set and a distal area belonging to the second set. Extensive overlapping occurs at least upon the main arteries, as we noted normal and degenerated fibres together on the lower part of the posterior tibial artery. The large degenerated peri-arterial trunks dissected in the coats of the femoral artery were obviously not by any means exhausted upon that artery. Such overlapping would help to explain the difficulty emphasized by Davis and Kanavel⁴ (1926) of understanding "how the interruption of the minor portion of the vascular sympathetic innervation from a local segment of an artery can affect the most distal portion of the extremity".

But if there is no clear regional difference (in a proximo-distal sense) between the supply areas of the two sets of nerves, one or two features in the present investigation suggest there may be a qualitative difference in areas. It has been noted above that a muscular branch of the femoral artery in Hunter's canal seemed to be completely innervated from the peripheral source. Degenerated fibres were apparently absent on the peroneal artery, which is usually an almost purely muscular vessel: its nerve-supply also was derived from the peripheral nerves. On the other hand, the posterior tibial artery, a mixed muscular and cutaneous trunk, showed both degenerated and normal nerve-fibres. The suggestion is therefore put forward that the incoming nerves to the vessels in the limb may be specially concerned with the vascular supply to the muscles. It cannot be entirely so concerned, however, as Kramer (1914) has demonstrated fine accessory nerves running to the digital vessels in the hand. It is well known that the blood-vessels in the muscles themselves, down to the capillaries, receive nerve-fibres from the ordinary muscular nerves. It may be recalled that the blood-vessels in the muscles and of the skin show differing physiological reactions. Hoskins, Gunning, and Berry⁵ (1916) have shown that adrenalin causes active vasodilatation in the muscles of the dog's leg, but vasoconstriction in the cutaneous vessels of the leg. Admittedly it is a matter of discussion how far the action of adrenalin on blood-vessels is mediated through the vasomotor nerves, but the striking difference of response in the muscular and cutaneous vessels calls for further investigation of any suggested difference in their nerve-supply.

The chief result of this investigation is to emphasize once again the dual paths of the vascular nerves to the limbs. It is obviously impossible to influence the whole of these by any proximal operation of peri-arterial

sympathectomy. Hence the preference of American surgeons—e.g., Davis and Kanavel (1926)—for cervical or lumbar sympathectomy operations in such cases is justified on anatomical grounds, apart from the desirability or otherwise, on general grounds, of the more severe type of operation then involved. Adson, at the Mayo Clinic, combines lumbar sympathectomy with peri-arterial sympathetic neurectomy of the common iliac arteries (Brown,⁶ 1926). Cohen⁷ (1925) admits the unsatisfactory result of peri-arterial sympathectomy in the thigh unless the femoral vein is ligated at the same time—which, of course, brings in a different method of treatment altogether; Brooke⁸ (1927) also recommends this combined procedure.

A minor issue, perhaps mainly of theoretical import, may be mentioned. In the classical operative-surgery class-procedure of ligaturing a main artery in continuity, the student is taught to work very strictly within the 'sheath' of the vessel. Thereby surrounding structures are safeguarded; but if ever such an operation is required in real life and peripheral vasodilatation in aid of collateral circulation is desired, the opportunity is missed of securing such a possible temporary effect in this direction as would be obtained by section of the peri-arterial nerves. As has been shown, they run in the separated 'sheath'.

SUMMARY.

1. The condition of the nerves in the main arteries was investigated in a leg amputated five weeks after the peri-arterial injection of alcohol into the sheath of the femoral artery.

2. All nerves running with the artery at the seat of the previous operation appeared to have degenerated distally, but an abundant additional supply running to the vessels lower down from the ordinary nerves of the limb then showed up in marked contrast.

3. This accessory nerve-supply arrives upon the vessels of the leg mainly in the popliteal space. The incidence of these accessory nerves was also checked by anatomical dissection in another limb.

4. The incoming nerve-twigs have a characteristic structure, being composed chiefly of small medullated fibres, with a few large medullated fibres and a fair number of non-medullated fibres.

5. It is suggested that these incoming nerves may be specially concerned with the blood-vessels to the muscles.

We wish to record our thanks to Professor T. H. Bryce, F.R.S., for accord-
ing us facilities for carrying out part of this investigation in the Anatomy
Department of the University of Glasgow.

REFERENCES.

- ¹ WOOLLARD, H. H., *Heart*, 1926, xiii, 319.
- ² POTTS, L. W., *Anat. Anzeiger*, 1914, xlvii, 138.
- ³ KRAMER, J. G., *Anat. Record*, 1914, viii, 243.
- ⁴ DAVIS, L., and KANAVEL, A. B., *Surg. Gynecol. and Obst.*, 1926, xlii, 729.
- ⁵ HOSKINS, R. G., GUNNING, R. E. L., and BERRY, E. L., *Amer. Jour. Physiol.*, 1916, xli, 513.
- ⁶ BROWN, G. E., *Jour. Amer. Med. Assoc.*, 1926, lxxxvii, 379.
- ⁷ COHEN, I., *Ann. of Surg.*, 1925, lxxxii, 704.
- ⁸ BROOKE, R., *Brit. Jour. Surg.*, 1927, xv, 286.

OSTEOPLASTIC CRANIOTOMY.***A NEW TECHNIQUE FOR THE RESECTION AND TURNING
DOWN OF BONE-FLAPS FROM THE SKULL.**

By LAMBERT ROGERS,

FIRST ASSISTANT IN THE SURGICAL UNIT OF THE WELSH NATIONAL SCHOOL OF MEDICINE, CARDIFF.

WITH AN INTRODUCTION

By PROFESSOR A. W. SHEEN. C.B.E.,

DIRECTOR OF THE SURGICAL UNIT.

INTRODUCTION.

THE technique of osteoplastic craniotomy set forth in this paper appears to me to benefit materially this branch of surgery. In the following account of his operation Mr. Rogers describes a form of circular saw which has made the cutting of bone-flaps from the skull a very easy and safe procedure.

In first outlining his idea to me the author explained that in Australia he had been impressed by the rapid and accurate way in which wooden planks fed to a running circular saw were cut, and there appeared to be no reason why a similar principle should not be applied to the skull provided that adequate protection for the brain and its membranes was assured. I agreed with the application of the principle, and hence the 'skull plough', a name which was suggested because the finished appliance looked rather more like a disc plough than a timber-cutting saw. I have now seen the instrument in use on many occasions and am impressed with its simplicity and effectiveness in the performance of osteoplastic craniotomy. In a leading article on cranial incisions in the *British Medical Journal* for Feb. 25, 1928, we read, "The ideal incision is that which makes the operation easy. for what is easy is generally safe." Lambert Rogers's 'skull plough', in appearing to fulfil these criteria, possesses advantages over other mechanical craniotomes which I have seen in use, and over such devices as Gigli saws.

It will be found that this paper is more than an account of a new craniotomy and the method of using it. It embodies the result of much experimental work, not only in the methods of cutting bone—and particularly the skull with its tables and intervening diploë—but on the way in which fractures occur at the base of bone-flaps. It is well known that the difficulty in performing osteoplastic craniotomy is frequently not the cutting of the flap but the turning of it down. The author has contributed experimental observations to this subject which appear to throw much light on the question and to simplify the procedure.

* From the Surgical Unit of the Welsh National School of Medicine, the Royal Infirmary, Cardiff.

OSTEOPLASTIC CRANIOTOMY.

"Surgical technique is the most worthy and least regarded nowadays of all the essentials of good work, yet it is the one that most surely crowns our efforts with success."—*Lord Moynihan*.

Historical.—Although opening the skull is such an ancient operation that Velpeau remarked, "*L'origine de la chirurgie crânienne se perd dans la nuit des temps*",¹ and examples of neolithic trephining, dating in all probability from before 2000 B.C.,* can be seen in many museums of anthropology, temporary resection of part of the skull by the formation of flaps comprised of its whole thickness—osteoplastic craniotomy—is of quite recent origin, having first been performed on a patient towards the close of the year 1889. Prior to that date the only form of temporary resection consisted of attempts—a few of which were successful—to replace the bone discs removed by trephining and in this way to close the skull completely at the conclusion of the operation.²

In 1864 Léopold Ollier³ of Lyons suggested lifting up a piece of the thickness of the skull adherent to a flap of soft parts. Two years before that date flap operations had been performed on animals by Julius Wolff⁴ in Berlin. But it was not until a quarter of a century after Wolff's experimental work and Ollier's suggestion that osteoplastic craniotomy was first undertaken on a living human subject. On Nov. 23, 1889, Wagner, of Königshütte, reported⁵ a case upon which he had performed the operation which has since come to be known by his name. In his paper, "*Die temporäre Resektion des Schädeldaches an Stille der Trepanation*", he described the operation which on Oct. 3 of that year he had performed upon a labouring man, age 27, who had been rendered unconscious by a coal-truck hitting him on the side of the head, and who two days after the accident had developed signs of hæmorrhage from the left middle meningeal artery. Wagner had previously experimented upon cadavers with the object of producing a bone-flap attached at its base to the soft parts so that the flap could be turned down, a reasonably large area of the brain exposed, and the flap subsequently replaced. He now applied this operation to his patient. A large bone-flap was chiselled out and turned down with its base still attached to the scalp, the extradural blood-clot was removed, the bleeding meningeal artery secured, and the flap replaced and sutured in position. Wagner's patient died twenty-four hours later, but the operation had introduced a new technique for the surgical exposure of the brain and its investing membranes, and it was not long before other and more successful attempts were carried out.

Methods of Performing the Operation.—Although some surgeons were hesitant to adopt the procedure, the advantages of osteoplastic resection of the skull were soon realized by others, who directed their attention to the best method of performing the operation. Wagner had used a hammer and

* Dr. Wilson Parry (*Jour. Brit. Archæol. Assoc.*, 1916, March) believes that the operation was performed in the Carnac Epoch of the Neolithic Period, which probably ended in North-Western Europe about 2000 B.C., and Paul Broca (*Bull. Soc. Anthropol.*, 1876) gives sound reason for thinking that it was performed even before this, viz., in the Campigny or Early Neolithic Period.

chisel, but in his paper he sounded a note of warning against hammering owing to the possibility of producing injury to the brain.* He stated, "I only use small strong chisels and elevators, but perhaps some form of circular saw could be constructed which could be set in motion after the manner of a dentist's drill with which it would be possible to work more quickly and accurately." Circular saws had already been introduced into cranial surgery, but at this time Wagner was apparently unaware of them, and it was not until his paper was in print that he added a footnote to the effect that Richter had told him of a saw after the type he had suggested, constructed by Messrs. Collin, of Paris, and used by Péan and Ollier.

Circular Saws for Craniotomy.—Before the appearance of Wagner's "Die temporäre Resektion", however, our great pioneer of cerebral surgery, the late Sir Victor Horsley, had realized the desirability of having a mechanical method for rapidly cutting through the cranial bones, and his biographer, Stephen Paget,⁶ writes that "so early as 1887 he added to the use of the trephine and the bone-cutting forceps the use of a miniature circular saw driven by a Bonwill's surgical engine" (Fig. 128).

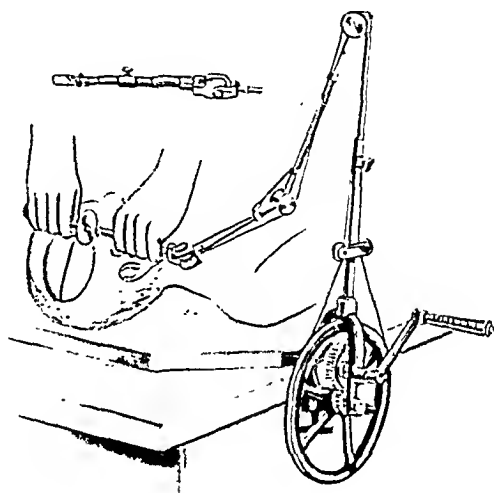


FIG. 128.—Sir Victor Horsley's circular skull saw.
(By the courtesy of Messrs. Mayer & Phelps.)

About this time also Macewen in Glasgow was experimenting with a circular saw for craniotomy, but neither Horsley nor Macewen performed osteoplastic operations, nor did they find circular saws suitable for cranial work. It was not long before Horsley abandoned his, and in 1893 we find Macewen cautioning against the use of circular saws for opening the skull.⁷

In Paris, in 1897, Doyen introduced a circular saw for cranial surgery (Fig. 129),† and in the same year, in New York, William Waldo Van Arsdale, Adjunct Professor of Surgery at the Polyclinic, published a paper entitled

* Gorodiski (*Ein Vorschlag zur Trepanation*, Erlangen, 1893), commenting upon this, states that for this reason the use of hammer and chisel for opening the skull was deprecated by Galen about A.D. 200.

† I am indebted to Messrs. Mayer & Phelps for kindly lending me the saws from which Figs. 129 and 130 are made and for kindly writing to Messrs. Collin, of Paris, about Doyen's saw. The following reply was received: "Nous avons l'avantage de vous faire connaître que la scie du Dr. Doyen a été construite par nous et présentée à la Société de Chirurgie vers l'année 1897."

"Remarks on Temporary Resection of the Skull by a New Method, with a Report on Three Successful Cases".⁸ This new method consisted of the use of a specially constructed concavo-convex disc saw (*Fig. 130*), which Van Arsdale had presented to the New York Surgical Society the year previously, in the October of which he had also published an account of the instrument in the *Annals of Surgery*.⁹

Horsley's, Macewen's, Doyen's, and Van Arsdale's circular saws were all driven by surgical engines connected to their driving spindles by flexible cables; but apparently all these saws were failures, for not one remains in

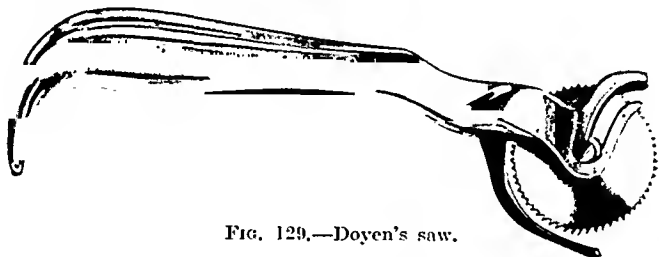


FIG. 129.—Doyen's saw.

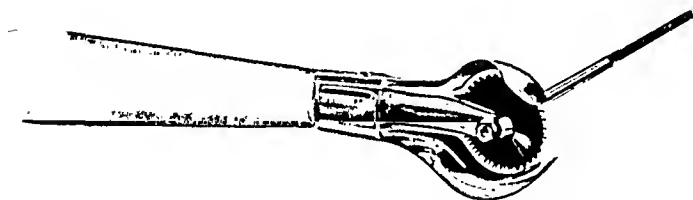


FIG. 130.—Van Arsdale's saw.

use to-day. Thus, for performing osteoplastic craniotomy, circular saws as constructed towards the end of the nineteenth century did not prove as useful as Wagner had hoped they might do.

Gigli Saws.—In 1894 a most useful instrument had been introduced by Leonardo Gigli, of Florence,¹⁰ and the flexible roughened wires, now universally known as Gigli's saws and still in wide usage, very soon became popular: in action these saws resemble the old chain saws* which were introduced during the eighteenth century for cutting long bones.

Gigli saws are too well known to need detailed description here, and it will be generally conceded that cutting bone-flaps from the skull by their means is a very safe procedure, but a somewhat laborious and lengthy one, since a number of trephine or burr holes are necessary, the passage of the saws along guards introduced between these holes is tedious, and the wires frequently jam or break.

Mechanical Craniotomes.—From time to time mechanical craniotomes, many of which have been most ingenious and highly complicated machines, have been introduced into cranial surgery with varying claims as to the

* Good examples can be seen in the Instrument Museum at the Royal College of Surgeons in Lincoln's Inn Fields.

advantages they possess in facilitating the performance of temporary resection of the skull. As examples of recent instruments, reference may be made to those of Jentzer, of Geneva,¹¹ De Martel, of Paris, and H. S. Souttar, of London.¹² There are disadvantages, however, appertaining to most mechanical craniotomes: some cut wide grooves, some concuss the patient, others tend to damage the meninges and the brain, while still others necessitate making holes in the centre of the bone-flap or are so complicated in their construction that it is conceivably quicker to open the skull with burrs and Gigli saws than to assemble and use the craniotomes. Further, most of these instruments require a good deal of preliminary handling and practice before it is justifiable to use them in the operating theatre.

The Problem of How to Improve the Gigli-saw Technique.—Although Sir Percy Sargent has developed a rapid method of cutting bone-flaps with drills and burrs, the use of a butcher's saw, and a few taps with a hammer and chisel, this technique obviously requires long practice and needs the hand of a master, and the first problem which still faces the great majority of surgeons who wish to expose the brain and its membranes through a replaceable bone-flap is how best to cut the flap with the greatest safety to the patient and the least distress to the surgeon. With the belief that the circular-saw method suggested by Wagner as a means of improving the technique of his temporary resection operation forms the solution of this problem, and despite the fact that this method of craniotomy had been unsuccessfully tried by many surgeons, including even Horsley and Macewen, some experimental work was undertaken with the use of circular saws.

An Albee's saw can, of course, be used very effectively, but this necessitates a number of openings into the skull and the passage of guards between them, and hence this technique proves very little, if at all, better than that in which Gigli saws are used. Attempts were therefore made to produce a simple craniotome, working on the circular-saw principle, and not requiring the preliminary passage of meningeal guards or the production of numbers of trephine or burr holes.

Examination of the circular saws which from time to time have been unsuccessfully employed for cranial surgery, and to some of which reference has already been made in this paper, reveals the fact that failure in each case has apparently been due to the difficulty of engaging a power-driven saw with the tables of the skull, while at the same time protecting the brain and the meninges from injury and maintaining an accurate directional and mechanical control over the instrument. This difficulty, however, was overcome by the production of an electrically driven disc-saw capable of accurately controlled biplanar movement, that in the vertical plane being towards and away from a flat guard which also serves as a separator to free the dura mater from the inner table of the skull. (*Figs. 131-134.*)

The Circular Saw adapted for Osteoplastic Craniotomy.—The instrument which, from its superficial resemblance to a disc plough or cultivator, has been called a 'skull plough' consists essentially of three parts: (1) A pistol grip carrying the saw; (2) The guard and separator for the dura mater; and (3) A circular saw measuring $1\frac{3}{8}$ inches in diameter, driven in a clockwise direction by a flexible cable from an electro-motor. The saw is capable of

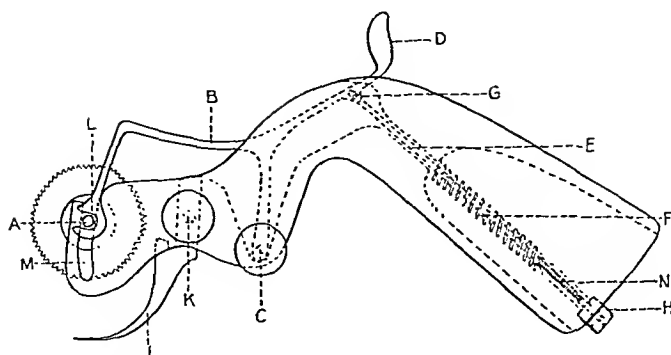


FIG. 131.—The 'skull plough': machine drawing—side elevation. A, Driving shaft of saw; B, Lever by which motion in a vertical plane is imparted to the saw; C, Fulcrum—a screw with milled head—from which the lever, B, acts; D, Thumb-push; E, Rod connected to lever, B, at G and carrying a thread, N, on which a milled screw, H, turns, controlling the degree of compression of a spiral spring, F; K, Screw with milled head fixing the dura mater guard, J, in position; L, Bearing for driving shaft of saw, moving through slot, M.

Note.—By unscrewing C, H, and K, the components of the instrument are easily taken apart for cleaning. The screw, K, permits angled guards to be inserted in place of J when it is desired to bevel the bone cut.

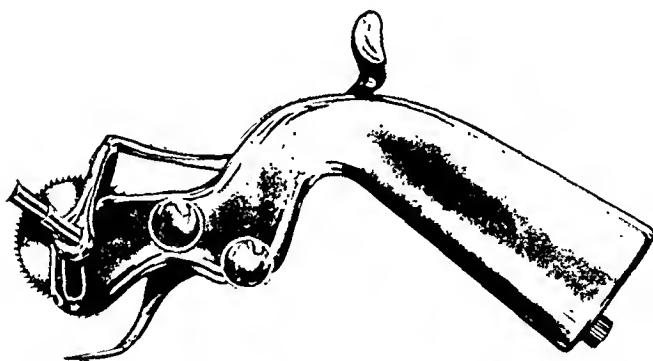


FIG. 132.—The 'skull plough': side view.

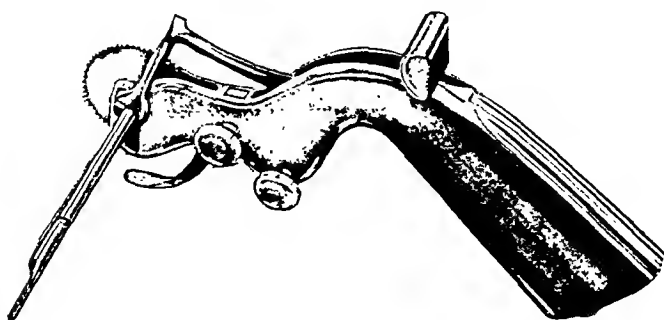


FIG. 133.—The 'skull plough': view from above and to the side.

movement in a vertical plane towards and away from the dura guard by pressure upon a thumb-piece against a spring which normally keeps the saw at the highest point of its vertical excursion. i.e., the maximum distance from the guard. The pressure on the thumb-push reduces this distance until, at its lowest point, the saw just clears the guard. The saw is capable also

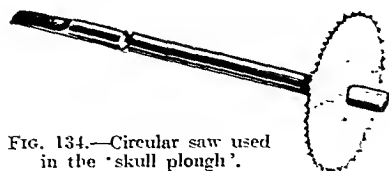


FIG. 134.—Circular saw used in the 'skull plough'.

of movement in a horizontal plane by pressure forwards of the pistol grip, depression of which with the ball of the thumb also keeps the dura guard in close apposition with the inner table of the skull. These several features are illustrated

FIG. 135.—Method of using the 'skull plough' (partly diagrammatic). The soft parts have been divided down to the bone, the openings in the bone made, and the guard of the plough introduced through one so as to lie between the dura mater and the bone. The operator's right thumb is pressing the thumb-push downwards and forwards and engaging the saw, while the fingers of the same hand are around the butt of the instrument which, being depressed, keeps the dura guard closely applied to the inner table of the skull. The left hand steadies the driving spindle.



in Figs. 131-134.* The motor develops about one-tenth of a horse-power.

The Technique of Cutting and Turning Down Bone-flaps from the Skull.
—The position and shape of the proposed bone-flap having been marked out, the soft parts are cut through along one limb of the flap, a heavy knife

* This instrument was demonstrated on July 24, 1929, to the Surgical Section at the Annual Meeting of the British Medical Association in Manchester, and has been made for me by Messrs. Mayer & Phelps, London, to whom I am much indebted for the patient way in which they allowed me to make alterations as the instrument evolved. I am also much indebted to Mr. P. G. Phelps of that firm for assisting me with certain mechanical problems involved in surgical instrument manufacture.

being used, and the tissues divided down to and including the pericranium (*Fig. 135*). While this incision is being made the hands of an assistant compress the edges of the wound and so control bleeding. Skin cloths are next fastened along the edges of the incision, using large Michel clips (No. 16)

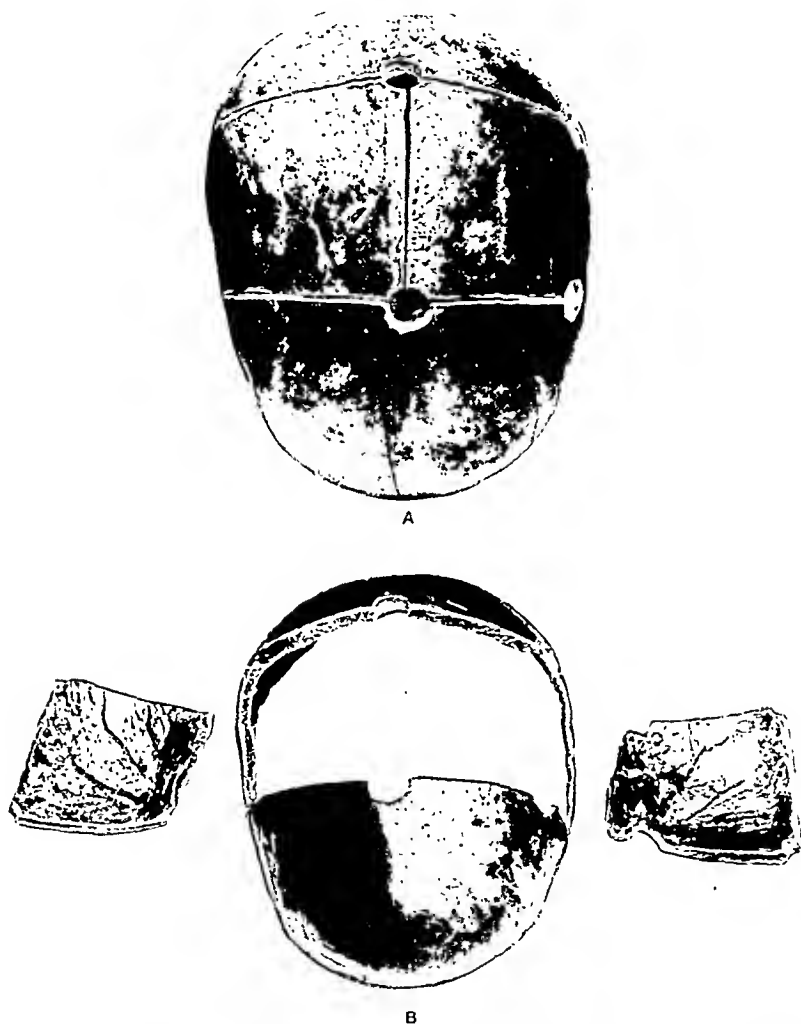


FIG. 136.—Experiment to show the way in which bone-flaps fracture. A, Two flaps of equal size and in corresponding position have been cut on the skull. The sides of the flap on the right terminate as saw cuts, those of the flap on the left as small circular holes (trephine or burr openings). B, Fracture of the two flaps has been produced. Note the comparatively straight fracture line of the flap on the right, and the jagged, irregular line between the trephine openings. This experiment has been repeated on numerous occasions and the results are consistent.

and taking a firm hold of the epicranial aponeurosis so that this is drawn outwards towards the cloths, and hæmorrhage is in this way completely arrested. The other two limbs of the flap are similarly dealt with, and two small ($\frac{3}{8}$ in.) trephine or burr holes are next made, one at each of the

meeting-points of the sides of the flap, the pericranium in this region being pushed away with a Farabeuf's raspatory, prior to drilling. The guard of the 'skull plough' is next passed through one opening, the saw being at the position of maximum vertical height, which is such that the thickest skull can be cleared, and the guard is made to lie between the dura and the inner table of the skull, and directed in the line of the proposed section of the bone. The pistol grip is now depressed so that the guard is kept closely applied to the inner table, and the saw, which is being rapidly rotated by the flexible drive from the motor, is engaged by gradually depressing the thumb-push, while the whole 'plough' is gently and evenly propelled along the desired line of section. To stop cutting, it is only necessary to relax the pressure of the thumb upon the thumb-push, when the saw, still of course rotating, rises out of the groove which it has been cutting.

Engaging the running saw with the bone is somewhat akin to 'letting in' the clutch of a motor-car, and must be gentle and gradually progressive to prevent the load becoming suddenly too great and the engine stalling. If this precaution is taken, this type of circular-saw craniotomy has been found to work smoothly and rapidly, and the three limbs of a large bone-flap can be cut in a few minutes. Also, by using angled meningeal guards the flap can be cut with bevelled edges, and very accurate apposition effected when at the end of the operation it is desired to replace the bone. By cutting the bone-flap so that two of the limbs

converge towards the base (*Fig. 135*), it has been found that the flap can be fractured easily at its base and that it breaks along an approximately straight line joining the termination of the saw cuts (*Figs. 136, 137 B*). This line of fracture tends to be remarkably regular,* and more so than when trephine or burr holes mark the ends of the line along which the bone is broken, as is necessary in using the Gigli-saw technique (*Fig. 136*).



FIG. 137.—Osteoplastic flap cut from a cadaver with the 'skull plough'. A, Flap in position; B, Flap turned down. Note the straight line of fracture at the base of the flap.

* An explanation of this has been given me by my colleague, Mr. J. E. Barlow, Lecturer in Engineering in Cardiff College, who points out that the concentration of stress around the trephine or burr openings would be much greater than around the ends of saw cuts.

Before applying this technique to a living subject, experiments were undertaken with the instrument in the dissecting-room* (*Fig. 137 A, B*), and it was not until June 3, 1929, some six months after designing the 'skull plough', that osteoplastic craniotomy on a living patient was first performed with it. It has now been used upon a number of cases under either general or local anæsthesia (*Figs. 138, 139*), and has proved so advantageous for performing Wagner's operation that it appears to fulfil his prediction as to the value of a circular saw for this purpose. A recent case will illustrate this:—

A well-developed farmer, age 33, was admitted to the Royal Infirmary in a very restless condition of semiconsciousness following a blow on the left side of the head with a ploughshare. Intracranial tension was kept reduced by four-hourly rectal injections of 30 per cent magnesium sulphate. There was no lucid interval followed by coma, but three days after his admission right monoplegia developed and soon progressed to hemiplegia. The systolic blood-pressure was only 90 mm. of mercury.



FIG. 138.—Patient six days after production of an osteoplastic flap by the technique described.

Five days after the accident, under chloroform-ether-oxygen anæsthesia, an osteoplastic flap, with its base beneath the temporal muscle, was turned down over the left motor area, a linear fracture traversing the parietal and squamous temporal bones was exposed, and 83 grm. of extradural blood-clot resulting from torn anterior branches of the middle meningeal vessels were removed. The bone-flap was cut with the 'skull plough'. Following the evacuation of the blood-clot, the flap was replaced and sutured in position. The systolic blood-pressure at the end of the operation was still 90 mm. of mercury. The patient made a complete recovery, power in the leg returning first, followed by that in the arm. (*Fig. 138.*)

This example of left middle meningeal hæmorrhage resembles in many respects that upon which Wagner in 1889 was the first to perform osteoplastic

* I am indebted to Professor C. M. West for giving me facilities to perform these experiments.



Lateral view.



Antero-posterior view.

FIG. 139.—X-ray appearances after replacing osteoplastic flap cut with 'skull plough'
The patient was a woman, age 52, with Jacksonian epilepsy.

craniotomy, using a chisel to cut his bone-flap, while suggesting that some form of surgical saw might be constructed to serve the purpose better. It will be remembered that Wagner's case was one of left middle meningeal hæmorrhage occurring in a labouring man of 27.

Advantages of the 'Plough' Technique.—The advantages claimed are:—

1. Rapidity of execution. Although in some clinics there would appear to have developed a surgical fashion of working very slowly at intracranial operations, sometimes prolonging them to a remarkable length of time, which must be a severe test of the stamina of both patient and surgeon, there seems to be little in favour of this attitude, and much against it. The shorter the time during which an operation wound is open, the fewer the chances of infection and the sounder the healing when the wound surfaces are approximated. Time-saving, therefore, must obviously be of importance provided it can be effected without danger to the patient.

2. When cutting the osteoplastic flap there is absolutely no danger of injury to the meninges or the brain, and thus the technique is a safe one.

3. There is an absence of any concussing force liable to produce cerebral contusion, such as occurs in the 'punching-out' type of craniotomes.

4. There is no need, as with some craniotomes, to trephine the centre of the proposed flap. A central opening in the bone is obviously a disadvantage if a flap is being turned down over a vascular meningeal tumour with overlying changes in the bone, since there may be troublesome hæmorrhage during the cutting of the flap.

5. The grooves made in the bone are narrow, and the flap can be cut with one or all of its edges bevelled so that it can be accurately replaced at the conclusion of the operation.

It is believed that the accurately controlled biplanar action of the 'skull plough' constitutes a new principle in the use of circular saws for surgical work, but the instrument is essentially so simple that it is surprising it has not been devised before. Although at first sight, and to those who have not seen the 'plough' working, it may appear rather complicated, it is not so really, and its successful action appears to depend on its essential simplicity. Lord Moynihan has said that "in the craft of surgery the master word is simplicity",¹³ and the technique of osteoplastic craniotomy as here set forth has been evolved with this ideal in view, the 'skull plough' being the result of attempts to produce the simplest and at the same time a perfectly safe method of adapting the circular saw to work in the way in which Wagner originally suggested osteoplastic craniotomy should be carried out.

CONCLUSIONS.

An attempt has been made to solve the problem of how to improve the technique of performing Wagner's operation of osteoplastic resection of part of the skull. It is believed that this problem has been solved by adopting Wagner's original suggestion to cut the bone with a circular saw, an instrument having been produced involving a principle which surmounts the difficulties that are inherent in the circular saws which

have previously been tried for this purpose, and which have failed because of these difficulties.

The 'skull plough', in facilitating the performance of the Wagnerian operation, adapts the circular saw to craniotomy in a manner which enables a bone-flap to be formed in a few minutes with both accuracy and absolute safety. Moreover, this bone-flap can be turned down easily—fractures along a straight line constituting its base—and at the conclusion of the operation can be accurately replaced and sutured in position.

REFERENCES.

- ¹ CHIPAULT, A., *Chirurgie opératoire du Système nerveux*, 1894. Paris : Rueff.
- ² OLLIER, L., *Traité des Resections*, 1891. iii. Paris : Masson.
- ³ OLLIER, L., "Note on a Method of Trephining", *Edin. Med. Jour.*, 1895, xl, 633 (quoted by J. M. Cotterill).
- ⁴ WOLFF, J., "Zur temporären Resektion des Schädeldaches", *Zentralb. f. Chir.*, 1890, Jan. 4, 1.
- ⁵ WAGNER, W., *Ibid.*, 1889. Nov. 23.
- ⁶ PAGET, S., *Sir Victor Horsley : A Study of his Life and Work*, 1919. London : Constable.
- ⁷ MACEWEN, Sir W., *Glasgow Med. Jour.*, 1893, i, 307.
- ⁸ VAN ARSDALE, W. W., *N. Y. Polyclinic Jour.*, 1897, 27.
- ⁹ VAN ARSDALE, W. W., *Ann. of Surg.*, 1896. xxv, 465.
- ¹⁰ GIGLI, LEONARDO, *Zentralb. f. Chir.*, 1894, xxi, 409 ; 1897, xxiv, 783 ; 1898, xxv, 425 ; *Sperimentale* (Sez. Chir.), 1895, 1.
- ¹¹ JENTZER, A., *Report of Seventh Congress of the International Society of Surgery*, Rome, April, 1926.
- ¹² SOUTTAR, H. S., *Brit. Med. Jour.*, 1928, i, 295.
- ¹³ MOYNIHAN, Lord, *Abdominal Operations*, 1926, i, 25. London and Philadelphia : Saunders.

PURPURA AS AN ACUTE ABDOMINAL EMERGENCY.

By HAMILTON BAILEY,

LATE SURGEON TO THE DUDLEY ROAD HOSPITAL, BIRMINGHAM.

PURPURA is a disease of great interest and importance to the surgeon. It forcibly intrudes into his diagnostic arena; on rare occasions it is the cause of an intussusception; it may give rise to profound hæmaturia or alarming hæmorrhage from a mucous lining of the body; it is a condition which calls for blood transfusion, and evidence is accumulating rapidly that certain carefully selected cases are permanently benefited by splenectomy.

In the past purpura has been divided into various types: (1) Purpura simplex; (2) Purpura rheumatica or Schönlein's disease; (3) Purpura abdominalis or Henoch's purpura; (4) Purpura hæmorrhagica or morbus maculosus Werlhofii; (5) Purpura fulminans. These subdivisions are incomplete, confusing, and unnecessary. Variations in symptoms are due to the location of the hæmorrhages. On this account Morse and Stone plead that these names should be dropped, and that in future we should speak of one clinical disease—purpura, with hæmorrhages and symptoms in various situations.

In this communication it is proposed to consider purpura from the standpoint of an acute abdominal emergency.

The Diagnosis of the Rash.—Purpuric spots have many times been mistaken for flea-bites, thereby confusing an important diagnosis. In the case of the extremities this mistake is hardly forgivable, for the spots of purpura are always maximally aggregated on the extensor surface. When the trunk alone is affected the differential diagnosis is more complex. In doubtful cases the tourniquet should be applied.

The Tourniquet Test.—When a rubber catheter is tied rather tightly around the arm of a patient suffering from purpura, petechial hæmorrhages appear distal to the constricted area. The tourniquet should be left in position for three minutes.

Hæmorrhage into the Wall of the Small Intestine.—Extravasation of blood into the wall of the gut gives rise to peritonism. Subserosal hæmorrhages in particular interfere with normal peristaltic action and produce symptoms of intestinal obstruction.

Case 1.—A boy of 8 was admitted in a very collapsed condition, with a diagnosis of intussusception. He presented a typical picture of advanced upper-gut obstruction. The eyes were sunken, the tongue was dry and brown, the pulse poor. He was vomiting ceaselessly. The note from his doctor stated that there was an ill-defined lump immediately above the umbilicus, which was confirmed. On turning the patient on his side to make a rectal examination it was noted that the buttocks were covered with purpuric patches, some of which were the size of a two-shilling piece. Purpuric patches were seen also on the back, and particularly on the lobules of the ears. In addition a number of small spots on the extremities and the

abdomen were observed. The diagnosis of purpura was evident, but it was thought that there was a concomitant intussusception. The abdomen was opened, and a most astonishing picture was revealed. About four feet of jejunum was the colour of bright-red blood (*Fig. 140*), and on closer examination extravasated blood could be seen beneath the serosa. The whole of this area was heavy with blood. No intussusception or obstruction was found. The abdomen was closed. The patient was given rectal saline during the night. Calcium lactate was administered later. Recovery.



Fig. 140.—Case 1. Subserosal extravasation of blood in purpura.

Case 2.—H. A., age 21, was sent to hospital as acute intestinal obstruction. He stated that he had had acute abdominal pain for forty-eight hours. The pain was spasmodic in character, and he had vomited copiously and repeatedly. On the table beside him was a receiver containing bile-stained vomit which he had just ejected. On inquiry it was elicited that one year previously he had had a similar attack lasting three days, and he volunteered the information that on that occasion he thought he had passed blood in the motions. The temperature was 98° and pulse 68. Preparatory to an abdominal examination, while the nurse adjusted the bedclothes the patient grasped the bedrail above his head, and in so doing exposed a large bruise on his arm. This was fortunate, for an examination of the limbs showed a typical purpuric rash (*Fig. 141*). Deep tenderness in the epigastrium was the only physical sign on abdominal examination. The rectal examination was negative. The diagnosis of purpura with submucosal hæmorrhage into the jejunum was made, and it was resolved to open the abdomen if the symptoms continued. As it happened, the abdominal pain and vomiting subsided within an hour, and in a fortnight the rash had faded almost completely. Some months later the patient was again admitted with similar symptoms, and he stated that attacks of colic

and vomiting had recurred at weekly intervals. As the platelet count was low, after three weeks' rest in bed the spleen was removed after preliminary blood transfusion. Ten months later the patient wrote to say that he had been quite free from colic and was at work, although the rash appeared if he allowed himself to become constipated.

As can be culled from *Table I*, a hæmorrhage into a limited portion of intestine often gives rise to a palpable tumour. Sometimes the abdominal symptoms precede the rash; in a case of Gracic's the rash came out eleven days after the onset of the abdominal symptoms, and several days have elapsed



FIG. 141.—*Case 2.* Showing the rash and the bruise which called attention to the condition. The patient appeared to be suffering from acute intestinal obstruction when first admitted.

in the cases reported by Osler, MacGillivray, and others. Little wonder, then, that surgical clinicians sometimes diagnose acute intestinal obstruction, intussusception, mesenteric occlusion, or some other intra-abdominal catastrophe normally requiring immediate operation, and open the abdomen only to find a collection of blood in the wall of the small intestine.

Several observers have likened the affected segment to stiffened gut after an intussusception has been reduced, but I take it this alludes to what is felt on palpation. The only condition which *looks* somewhat like the bloody extravasation of purpura is mesenteric thrombosis. In the case of purpura in which I opened the abdomen the affected gut presented an appearance which may be reproduced exactly by injecting arterial blood under the serosa.

PURPURA AS AN ABDOMINAL EMERGENCY 237

Table I.—CASES IN WHICH LAPAROTOMY WAS PERFORMED AND HÆMORRHAGE INTO THE WALL OF THE INTESTINE FOUND.

AUTHOR	SEX	AGE	DIAGNOSIS	OPERATIVE FINDINGS	RESULT
1. Greig	M.	9	Intussusception (lump palpable)	Hæmorrhage into wall of small intestine	R.
2. Murray, reported by Sutherland	M.	5	Intussusception (lump palpable)	Hæmorrhage into wall of ileum near ileocaecal valve	R.
3. Cook	M.	12	Intussusception (lump palpable)	Hæmorrhage into wall of small intestine	D.
4. Pybus	F.	9	? Intussusception (no lump)	Hæmorrhage into wall of lower ileum	R.
5. Fitzwilliams	A child		Intussusception (lump palpable)	Hæmorrhage into wall of lower ileum	D.
6. Kennedy	M.	9	Intussusception (lump palpable per rectum)	Hæmorrhages into wall of small and large intestine	R.
7. Dive and Purkiss	M.	19	Intussusception (lump palpable)	Hæmorrhage into wall of small intestine	D.
8. Mills	M.	16	Chronic intussusception	Patches of œdema and hyperæmia along the small intestine	R.
9. Burrows	M.	11	Intestinal obstruction	Hæmorrhage into wall of ileum	R.
10. Hadley	F.	19	?	Hæmorrhage into greater part of wall of jejunum	R.
11. Molesworth	F.	15	Appendicitis	Hæmorrhage into wall of small intestine	R.
12. Machol and Sthamer	M.	13	? Appendicitis	Hæmorrhage into ileum. Multiple hæmorrhages under peritoneum over mesenteric glands	D.
13. Harrigan	F.	13	? Appendicitis	Hæmorrhage into wall of lower ileum	R.
14. Patterson, reported by Hunter	M.	50	? Perforated gastric ulcer	Extravasation of blood into wall of jejunum	D.
15. Widgery	M.	23	?	Hæmorrhage into wall of small intestine (ileo-ileostomy performed)	R.
16. Bailey	M.	8	Intussusception (lump palpable)	Hæmorrhage into wall of jejunum	R.

R. = Recovered D. = Died.

The sheen of the serosa is heightened; moreover, on gently touching the serosa it can be made to move on the underlying blood.

Table II.—INTUSSUSCEPTION COMPLICATING PURPURA.

AUTHOR	SEX	AGE	TYPE OF INTUSSUSCEPTION	OPERATIVE TREATMENT	RESULT
1. Vierhuff (1893)	M.	29	Ileocaecal	No operation	Intussuscepted portion of bowel passed per rectum
2. Sutherland (1896)	F.	7	Ileocaecal	No operation	D.
3. Lett (1908)	M.	3	(a) Ileocaecal (b) Ileo-ileal (recurrence)	(a) Intussusception reduced (b) No operation	D. 9 days after reduction of first intussusception
4. Morse and Stone (1909)	F.	7	Ileocolic	Intussusception reduced	R.
5. Collinson (1910)	M.	4	Ileo-ileal	Intussusception resected	R.
6. Robinson (1910)	M.	5	Ileo-ileal	Intussusception resected	R.
7. Tonking (1910)	M.	5½	Ileo-ileal	Intussusception reduced	R.
8. McKechnie (1911)	?	2	Ileo-ileal	Intussusception reduced	R.
9. Gara (1912)	M.	5	Ileocaecal	No operation	D. Intussusception found at autopsy
10. Barling (1913)	M.	4	Ileocaecal	Intussusception reduced and affected bowel resected	R.
11. Lederer (1913)	M.	2	?	No operation	Intussuscepted portion of bowel passed per rectum
12. De Lavergne and Guillemin (1927)	M.	21	Ileo-ileal	Intussusception resected	D.
13. Caizergues (1929)	F.	3	Ileo-ileal	Intussusception reduced	R.
14. Ballin and Morse (1930)	M.	31	Ileocaecal	Intussusception reduced	Operation successful, but died of hæmaturia 2 months later

Acute Intestinal Obstruction Following the Subserosal Extravasation of Blood.—

H. A. Donaldson's case.—The patient, a boy of 11, had an attack of purpura with acute abdominal symptoms, and passed blood per rectum. During convalescence he had a sudden attack of acute intestinal obstruction. Laparotomy was performed forthwith. About five feet above the ileocaecal valve was a tubular constriction of

the intestine half an inch in length. All other structures were normal, and there was no Meekel's diverticulum. Lateral anastomosis was performed and the patient recovered.

Donaldson considers—very rightly, I think—that the constriction was the direct result of the extravasation of blood. Cicatrization about the intestinal hæmatoma is a potential late complication of purpura with abdominal symptoms.

Purpura and Intussusception.—A submucosal hæmorrhage tends to produce an excrescence protruding into the lumen of the intestine; obviously, such an excrescence invites intussusception. Up to the time of Hugh Lett's classical paper in 1908 no case of intussusception following purpura had been submitted to laparotomy. Since that time nine cases so treated have been reported (*see Table II*). Of these, seven have recovered completely. In no fewer than four instances has it been necessary to resect the intussusception; three of these four patients recovered, which is a tribute to surgical progress. On the other hand, as the intussusception was found to be gangrenous or irreducible in nearly half the total number of cases submitted to laparotomy, one is driven to the conclusion that there had been undue delay in opening the abdomen.

Purpura with Intestinal Symptoms. Selecting Cases for Laparotomy.—To diagnose purpura before the rash has appeared is manifestly impossible, and laparotomy will occasionally, and quite rightly, be performed in those cases which mimic an acute abdominal emergency. To overlook a purpuric rash or to mistake it for flea-bite is an occasion for self-criticism, but the question with which we are principally concerned is what course to pursue when purpura is evident and a concomitant intussusception suspected. An ill-defined lump (*see Table I*) is but a feeble guide to the necessity for laparotomy, yet to miss an intussusception must be our constant fear. The pinnacle of misfortune in this respect was reached by Sutherland. He advised laparotomy in his first case, and an extravasation of blood was found. On the next occasion he persisted in medical treatment, to be rewarded by the demonstration of a gangrenous intussusception at necropsy. In spite of the rarity of intussusception, most of us will agree that it is better to explore. This would be universally accepted if the patient's exit had not been hastened by bleeding from the wound, as occurred in two of the reported, and probably in several unreported, cases. If laparotomy was followed in a day or two by blood transfusion, this reproach would be removed.

After-treatment.—All patients who survive an attack of purpura should be carefully followed up. In chronic or recurrent cases the advisability of recommending splenectomy should receive full consideration. As far as our knowledge goes at present the most valuable guide is the platelet count. To quote Colin and Lemann—"A diminished platelet count bears the same relationship to purpura as a fall of blood-pressure does to shock, or the thermometer to a febrile reaction. The platelets are the indicators." They indicate that the patient will, probably, be benefited by removal of the spleen.

General.—

REFERENCES.

- COLIN, I., and LEMANN, I. I., *Surg. Gynecol. and Obst.*, 1924, xxxviii, 596.
KIDD, F., *Proc. Roy. Soc. Med.*, 1927-8, xxi, 1105.

General, *contd.*—

- GRACIE, J., *Practitioner*, 1924, cxiii, 419.
 HENOCH, E., *Berl. klin. Woch.*, 1874, xi, 641.
 OSLER, Sir W., *Brit. Med. Jour.*, 1914, i, 517.
 MACGILLIVRAY, A. G., *Ibid.*, 1927, i, 279.
 SPENCE, A. W., *Brit. Jour. Surg.*, 1927-8, xv, 466.
 EVANS, W. HOWEL, *Liverpool Med.-Chir. Jour.*, 1929-30, n.s. i, 40.
 LAVALLEE, M. A., *Thèse de Paris*, 1911.
 CALMELS, C. M. F. X., *Ibid.*, 1902.

Purpura and Laparotomy.—

- BURROWS, H., *Brit. Jour. Child. Dis.*, 1904, i, 23.
 HADLEY, J. A., *Brit. Med. Jour.*, 1927, i, 720.
 DIVE, G. H., and PURKISS, K. N., *Jour. R.A.M.C.*, 1921, xxxvi, 382.
 HUNTER, W. K., *Lancet*, 1928, ii, 1327.
 MOLESWORTH, H. W. L., *Ibid.*, 1922, i, 943.
 GREIG, D. M., *Scot. Med. and Surg. Jour.*, 1908, xxii, 302.
 SUTHERLAND, G. A., *Brit. Jour. Child. Dis.*, 1904, i, 23.
 PYBUS, F. C., *Lancet*, 1909, ii, 1074.
 COOK, J. B., *Ibid.*, 1587.
 FITZWILLIAMS, D., *Rep. Soc. Study Dis. Child.*, 1907-8, viii, 320.
 HARRIGAN, A. H., *Med. Record*, 1918, xcii, 1033.
 KENNEDY, A. M., *Clinical Jour.*, 1928, lvii, 337.
 MACHOL, A., and STIANIER, E., *Med. Jour. S. Africa*, 1924-5, xx, 218.
 MILLS, G. P., *Lancet*, 1909, ii, 223.
 WIDGERY, F. W., *Brit. Med. Jour.*, 1930, ii, 249.

Purpura and Intussusception.—

- LETT, HUGH, *Brit. Jour. Child. Dis.*, 1908, v, 343.
 SUTHERLAND, G. A., *Pediatrics*, 1896, ii, 412.
 VIERHUFF, J., *St. Petersburg Med. Woch.*, 1893, x, 369.
 GARA, A., *Jahrb. f. Kinderheilk.*, 1912, n.f. lxxvi, 573.
 LEDERER, R., *Zeits. f. Kinderheilk.*, 1913, Orig. vi, 227.
 COLLINSON, F. W., *Lancet*, 1910, i, 716.
 ROBINSON, H. B., *Ibid.*, 1009.
 TONKING, J. H., *Ibid.*, 802.
 MCKECHNIE, R. E., *Canad. Med. Assoc. Jour.*, 1911, i, 1040.
 MORSE, J. L., and STONE, J. S., *Arch. of Pediatrics*, 1909, xxvi, 287.
 BARLING, SEYMOUR, *Brit. Med. Jour.*, 1913, i, 659.
 BALLIN, M., and MORSE, P. F., *Ann. of Surg.*, 1930, xci, 711.
 CAIZERGUES, J. F., *Nourrisson*, 1929, xvii, 99.
 DE LAVERGNE and GUILLEMIN, A., *Rev. méd. de l'Est.*, 1927, lv, 1.

THE MIXED TUMOURS OF THE SALIVARY GLANDS.

By D. H. PATEX,

ASSISTANT IN THE BLAND-SUTTON INSTITUTE OF PATHOLOGY, MIDDLESEX HOSPITAL.

INTRODUCTION.

THE salivary glands, more especially the parotid, but also to a lesser extent the submaxillary and other smaller glands, are the not infrequent site of an interesting series of tumours, which in this country are generally known as 'mixed tumours'. The tumours of this group show many variations of histological structure, and as a result we find applied to individual examples in the literature, in accordance with the particular appearance noted, such terms as 'myxoma', 'fibro-myxoma', 'fibro-myxo-endothelioma', 'endothelioma', 'adenoma', 'adenoid cystic epithelioma', 'chondroma', 'chondro-carcinoma', and 'cylindroma'. The confusion of terminology is aggravated also by lack of agreement on the derivation of the tumours. Among the numerous suggestions made, it has been argued that they are epithelial, that they are endothelial, that they are derived from sequestered collections of embryonic cells or from some portion of the branchial arches, and that they have a truly mixed origin. So much of the recorded work deals with this question of derivation that more practical issues have been rather neglected and obscured by a mist of hypothesis. A certain number of efforts, however, have been made to evolve a pathological classification which shall be of practical value in indicating to some extent the probable clinical course of a given tumour, and the prognosis after operation. Thus Wood¹ (1904) classified his cases into three groups: (1) Hard, fibrous, and acellular tumours; (2) Cartilaginous tumours; (3) Cellular tumours. The first two groups he regarded as relatively benign, and the last as relatively malignant, but no figures are given in support of these conclusions, and the classification is also unsatisfactory in other ways. Wilson and Willis² (1912) subdivided their cases in a similar manner. Of 9 cases of recurrence in 42 patients traced in their series, 5 were classified as being of the cellular 'sarcomatous' type, and 4 as of the 'fibrous' type.

From such work as has appeared on the subject, the general impression seems to be that there are two chief types of tumour: (1) A soft, highly cellular tumour liable to recur after operation; and (2) A harder acellular, fibrous tumour, operative removal of which will probably be followed by prolonged freedom from recurrence. The views of McFarland³ (1926), however, form a notable exception. As the result of a study of 90 cases, this observer came to the conclusion that no relation could be established between the histological and clinical features of parotid tumours, and that "the microscope, beyond showing that the lesion is a mixed tumour, is misleading rather than enlightening".

SCOPE OF THE PRESENT INQUIRY.

We are thus in the position of having to deal with a group of tumours the derivation of which is a matter of dispute, concerning the terminology of which there is no general agreement, and of which no satisfactory pathological classification has yet been determined. The main aim of the present inquiry was to see whether a histological classification could be evolved which would allow of some degree of correlation of the clinical and pathological features, or whether the two were entirely unrelated as claimed by McFarland. The objects of the paper are thus primarily practical, though at the same time the conclusions formed during the course of the study on such theoretical questions as the derivation of the tumours will be given, together with the evidence on which these conclusions are based.

Material and Method of Study.—The material consisted of 55 cases of 'mixed tumours' of the salivary glands treated in the wards of the Middlesex Hospital since 1919, and of the pathological material from 52 of these cases which was available for study in the Bland-Sutton Institute of Pathology. In addition, a certain number of fresh tumours removed outside the hospital have been examined. The pathological and clinical aspects of the study were at first kept quite separate, and it was only after the pathological investigations had been completed that the task of tracing the after-histories of the patients was begun. The after-histories in the case of the patients traced are correct to the end of July, 1929.

The results of the investigation may be conveniently considered under the following headings: (I) *The histological appearance of the tumours*; (II) *Their clinical course*; (III) *The correlation of the clinical and histological findings*.

I. THE HISTOLOGICAL APPEARANCE OF THE TUMOURS.

It will be convenient to begin by describing the microscopical appearances of a 'typical' mixed tumour. Such a tumour is composed partly of collections of cells, partly of myxomatous tissue, the two elements being intermingled in varying proportions in different parts of the same tumour, while the whole tumour is as a rule enclosed within a well-defined fibrous-tissue capsule (*Fig. 142*). A characteristic microscopical field is illustrated in *Fig. 143*. One sees collections of cells with small dark angular nuclei arranged in irregular masses and branching columns, while between the cellular areas is a myxomatous tissue. There is usually no abrupt demarcation between the cellular and the myxomatous areas, the histological appearances suggesting very strongly that the myxomatous areas are

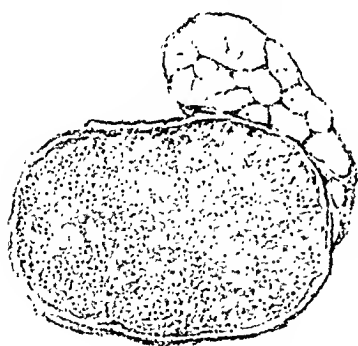


FIG. 142.—Typical encapsulated mixed tumour from the submaxillary gland.

being derived from the cellular areas. Though the collections of cells for the most part show no tendency to arrange themselves in any definite structural

formation, yet in nearly all tumours may be found areas such as those illus-



FIG. 143.—Typical parotid tumour showing masses and columns of cells containing small dark angular nuclei, and intermediate myxomatous areas with similar cells. ($\times 60$.)

trated in *Fig. 144*, in which an alveolar or ductal arrangement is reproduced. In different tumours greater or lesser numbers of cells will show this arrangement, while in some—as, for example, the one from which the illustration was taken—the process may take place to such an extent that an appearance is produced not unlike that found in a fibro-adenoma of the breast. On the other hand, in some cases the cell masses have almost entirely disappeared, the greater part of the tumour being composed of myxomatous tissue. Such a tumour is illustrated in *Fig. 145*, and it will be seen that, apart from a thin layer of cells at the periphery, practically the entire tumour is myxomatous. We may thus say that a 'typical' mixed tumour is formed histologically of three elements in varying proportions: (1) Collections



FIG. 144.—A tumour from the parotid, in which the cells are tending to arrange themselves in alveolomatus formation. In between is the usual myxomatous tissue showing fibrillar condensation. ($\times 60$.)



FIG. 145.—Tumour which has undergone almost complete myxomatous change, a thin layer of cells at the periphery alone remaining. ($\times 40$.)

of cells without definite arrangement; (2) 'Adenomatous' areas; (3) 'Myxomatous' areas. Moreover, all gradations in structure between these three component elements may be observed, one area merging into another, and the histological appearances suggesting very strongly that all three elements are merely different manifestations of the same tissue.

Apart from the typical group, certain tumours stand out microscopically on account of their high cellular content. These highly cellular tumours are of various kinds. We have already noted, in dealing with the typical tumours, that the collections of cells may be either arranged diffusely without a definite formation, or well differentiated into structures resembling ducts and alveoli. The same applies to the highly cellular tumours. The first type to be recognized, therefore, may be regarded as a highly cellular counterpart of the tumour illustrated in *Fig. 143*. That is to say that its cells resemble

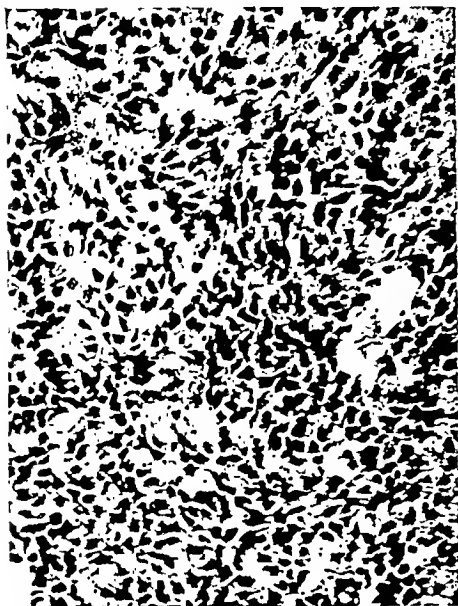


FIG. 146.—Cellular tumour in which the cells still retain the characteristics of those of a typical mixed tumour. This is the type of growth sometimes wrongly called 'sarcomatous'. ($\times 340$.)

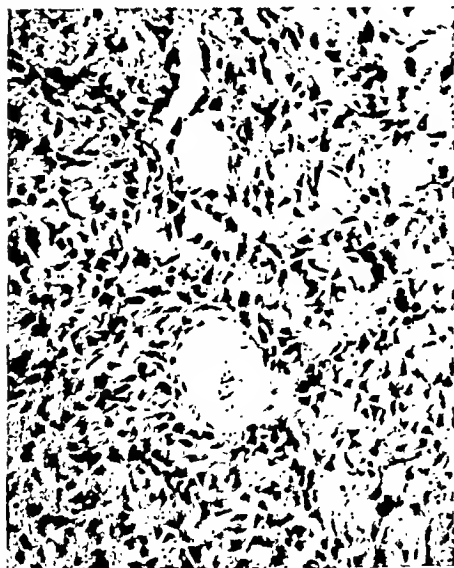


FIG. 147.—Same case as *Fig. 146*, showing immature tubule formation. ($\times 340$.)



FIG. 148.—Tumour showing a marked tendency to tubule formation. ($\times 60$.)

those of that tumour in structure, staining, and arrangement, but differ in that they are much more numerous and diffusely spread throughout the whole section. *Fig. 146* shows a tumour of this nature. This is the appearance which has given rise to the term 'sarcomatous type' employed by some authors. But even in these tumours, areas will be found in which attempts at immature tubule formation can be seen—*Fig. 147*, for example. For purposes of classification, tumours of this group may be termed "highly cellular and not well differentiated, but cells similar in type to those of a typical tumour".

On the other hand, certain highly cellular tumours are met with in which the cells, instead of being diffuse as in the previous type, are for the most part arranged in a well-marked alveolar or ductal formation. As a rule the

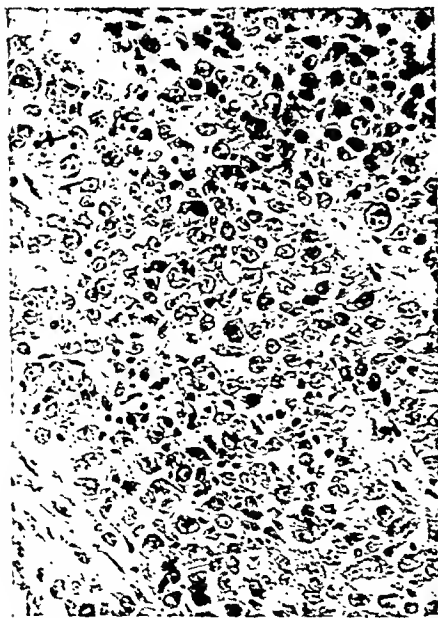


FIG. 150.—Portion of a rapidly growing cellular tumour, showing irregularity in size and shape of nuclei, and mitotic figures. ($\times 350$.)

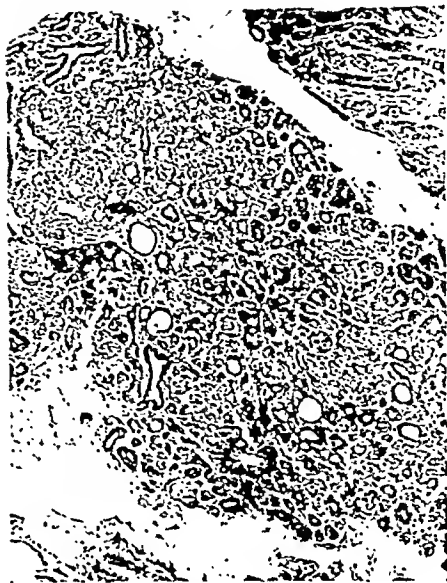


FIG. 149.—A portion of a cellular encapsulated tumour of the parotid, in which a high degree of differentiation into ducts and acini has taken place. ($\times 60$.)

process of differentiation stops short at the formation of simple tubules (*Fig. 148*), often containing a colloid-like secretion; but occasionally it progresses to the degree that ducts, acini, and an immature lobular arrangement can be recognized, the appearance closely resembling that of a normal functioning gland (*Fig. 149*). This is the type of tumour that constitutes the 'adenoma' of some authors. The point it is wished to emphasize here is that this highly differentiated appearance is merely the end-result of a process which may be recognized in lesser degree in the large majority of the mixed tumours. We may classify tumours of this group as "cellular but well differentiated".

Finally, a type may be recognized in which there is a high degree of cellularity, but in which, unlike the previous two tumours, the cells have lost their resemblance to those of a

typical tumour, and show evidence of rapid and disorderly growth in the form of irregularity and such nuclear changes as hyperchromatism and excess of mitotic figures. Such a tumour is illustrated in *Fig. 150*. This group is almost always poorly differentiated. For purposes of classification it may be termed "highly cellular, and not well differentiated, and cells showing irregularity and the nuclear changes of rapid disorderly growth". This group shades into that of the frank carcinomata. A consideration of where the boundary line between the two may be arbitrarily drawn will be left to a subsequent paper, but it may be here provisionally stated that, while a mixed tumour may under certain conditions infiltrate locally, it does not ordinarily metastasize, whereas the carcinomata soon tend to spread to distant parts, in the earlier stages by the lymphatics, but later by the blood-stream also.



FIG. 151.—Section to show the varieties in appearance which may be met with in the same tumour. Above and to the left is a highly differentiated area; below and to the right, strands of undifferentiated cells in a myxomatous stroma; and below and to the left, an intermediate stage. ($\times 40$.)

We thus have four groups into which the mixed tumours can be divided: (1) 'Typical' tumours; (2) Cellular, and not well differentiated, but cells similar in type to those of a typical tumour; (3) Cellular, but well differentiated; (4) Cellular, and not well differentiated, and cells showing irregularity and nuclear changes. Most tumours will fall quite readily into one or other of the above groups, but occasionally difficulty will be experienced, either because an intermediate type is met with—especially between (1) and (2)—or because of the varying appearances of different parts of the same tumour. For such cases as these the individual observer has to adopt an arbitrary standard.

The Derivation of the Mixed Tumours.—As already stated, the primary object of the present inquiry was to see whether a pathological classification could be evolved which would allow of some degree of correlation between the clinical and

pathological features of the mixed tumours, and it was not proposed to enter into any prolonged discussion on the question of derivation. But, while this series of tumours was being examined and the above classification evolved, the conclusion could not be avoided that one was dealing with the different manifestations of an epithelial tumour showing varying degrees of cellularity, and in various stages of differentiation. Since it is only on such a basis that the proposed classification is a rational one, it will be convenient at this point to turn aside for a moment from more practical issues and consider briefly the evidence for the epithelial origin of these tumours. First will be given the

evidence derived from the present study, and secondly that obtained from the literature.

We have already noted that an epithelial origin is suggested by the fact that in different tumours all gradations may be observed between an undifferentiated mass of cells, and a highly differentiated tumour with ducts, acini, and an attempted lobular arrangement. Occasionally one may meet with the whole range of gradations in the same tumour. The tumour illustrated in *Fig. 151* is an example. In the same microscopical field may be seen—above and to the left, a well-differentiated area; below and to the right, a 'typical' tumour containing collections of cells without definite arrangement and myxomatous tissue; and below and to the left, a stage intermediate between the two. An examination of this section left no doubt that it was the same type of cell occupying all these areas, a fact which the photograph also clearly shows. Again, in *Fig. 152* we see a

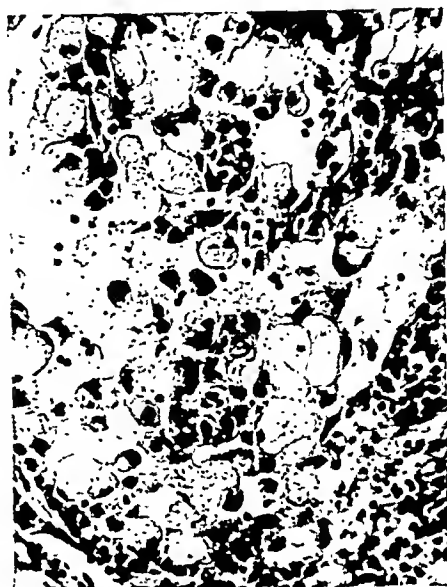


FIG. 152.—Cells from a parotid tumour distended with a granular secretion. ($\times 340$.)



FIG. 153.—A mixed tumour showing a structure formed of laminated keratin which closely resembles a cell-nest. ($\times 40$.)

tumour the cells of which show their origin from an epithelial secreting organ in the form of a granular secretion distending them almost to bursting-point. In *Fig. 153*, on the other hand, the cells are tending to differentiate rather in the direction of squamous epithelium, and we find in one place a structure formed of a laminated keratin-like material, which bears a close resemblance to a cell-nest. The evidence of the present study, therefore, all points to the conclusion that we are dealing with an epithelial tumour.

The idea that these tumours are epithelial in origin is by no means new, and much evidence in support of the theory has been adduced in the literature. At present it claims the support of a great number of pathologists, and is being accepted as the standard teaching of an increasingly large number of surgical and

pathological text-books. The chief stumbling-block in the way of a general acceptance of the epithelial theory in the past has been the presence in many of the tumours of areas of so-called cartilage. On this question Bland-Sutton⁴ has written: "A critical consideration of the histological features of the tissue called cartilage in parotid tumours convinces me that it is not entitled to this distinction"—an expression of opinion which the present study confirms. Krompceher⁵ (1900) regarded the salivary growths as of purely epithelial origin with peculiar mucoid and cartilaginous metaplasia, while Ehrich⁶ (1906) also demonstrated that the areas of so-called cartilage developed from the epithelial cells. In more recent times Fraser⁷ (1918) by means of a cresyl-violet stain, and Fry⁸ (1928) using nuclei-carmin, have been able to show that the cartilaginous appearance in certain areas is merely due



FIG. 154.—A sebaceous adenoma of the skin of the chest, showing histological features similar to those of the mixed tumours of the salivary glands. ($\times 40$.)



FIG. 155.—Another portion of the same tumour as seen in Fig. 154, showing pseudo-cartilage formation. ($\times 40$.)

to a fibrillar condensation of the myxomatous tissue with a resulting pseudo-capsular arrangement around the cells.

In the series under consideration the presence of areas morphologically resembling cartilage has been rare. In only three cases of the series was any difficulty experienced, and the majority of areas which on superficial examination resembled cartilage were on more careful examination obviously myxomatous. Further evidence in support of the above views is furnished by the finding of similar areas in other epithelial tumours in regions remote from the salivary glands. For example, in the tumour the microscopical appearances of which are illustrated in Figs. 154 and 155, and which was removed from the skin of the front of the chest, certain of the cellular lobules of which

the tumour was composed microscopically showed in the centre a clear hyaline material comparable to the 'cartilage' of the mixed tumours of the salivary glands. In *Fig. 156*, which is a microphotograph of a similar tumour removed from the soft tissues of the thumb of another patient, the resemblance to cartilage is even more striking. Both these tumours were probably adenomata arising in sebaceous glands, and both were quite superficial and encapsulated. In neither, therefore, would any sequestration theory be acceptable as explaining the origin of the 'cartilage', and the simplest and most obvious explanation is that this material, whatever its nature, is derived from the epithelial cells.

A position has thus been reached at which it is necessary to accept one of two views about this so-called cartilage. Either it is not cartilage at all, but a condensation of an epithelial-derived myxomatous tissue bearing a microscopical resemblance to cartilage; or, if it is true cartilage, then it comes from the epithelium by a process of metaplasia. To accept the latter view involves a rejection of biological tenets, but on this point Ewing⁹ has written: "However violently this interpretation may conflict with long-established views regarding tissue growth, it appears to be necessary to accept the facts so well attested by a close study of these tumours." Chemically there is no fundamental reason why such a change should not take place, since myxomatous tissue and cartilage are probably closely related. At any rate, it may be stated that the occasional demonstration of an area resembling cartilage is no valid objection to the epithelial nature of the mixed tumours of the salivary glands.

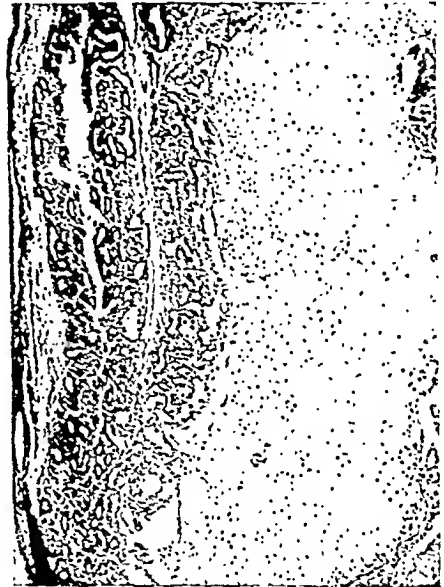


FIG. 156.—Sebaceous adenoma from the thumb with 'cartilage' formation as in the parotid.

Terminology.—If the epithelial origin of the mixed tumours is accepted, the question arises what they should be called. The term 'adenoma' might be used as denoting an innocent tumour of an epithelial secreting gland; but, in view of the rather special features of the salivary-gland tumours, it might be as well to retain the term 'mixed tumour', regarding it, however, not as referring to the derivation of the tumour, but as an expression of the varied histological appearances encountered.

II. THE CLINICAL COURSE OF THE DISEASE.

In this section the clinical features will be considered alone, without any attempt at correlation with the pathological findings, the latter task being left until the next section. In all there are 55 cases for analysis, in 52 of

which there was histological confirmation of the diagnosis; 3 cases are admitted in which no microscopical examination was made on account of their typical clinical characteristics. These 55 cases constitute all those treated at the hospital from the beginning of 1919 to the beginning of 1929. The results of the post-operative inquiry are correct to the end of July, 1929.

Region Involved.—The site of origin of the tumours was as follows:—

	CASES			
Parotid	38
Submaxillary	6
Palate	5
Other regions	5
(Lip, angle of mouth, face, tongue, sublingual gland.)				
Not known	1

In agreement with general findings, the parotid is much the commonest site for the tumours, though the submaxillary and palatal glands are by no means infrequently involved. No adequate explanation has been offered to account for the special predisposition of the parotid as compared with other salivary glands. The relatively large number of cases in the palatal region is interesting. Though cases of this nature have been recorded from time to time, it is only within recent years that it has been recognized that they probably constitute the commonest encapsulated tumours of this region. The submaxillary salivary gland is involved sufficiently often to render unnecessary the undue hesitation that is still seen in making a diagnosis of mixed tumour of this region. The tongue case is worthy of note on account of its rarity. As for the tumours in the remaining regions, it will be noticed that they all tend to be grouped close to the orifice of the mouth.

Recurrence after Operation.—Dealing, as the present series does, with cases that have been treated at various times between the beginning of 1919 and the beginning of 1929, the period during which the liability to post-operative recurrence has existed varies from ten years in the earlier cases to a few months in the later. In some of the cases much more than ten years' post-operative history was available, owing to the fact that the patients had had previous operations, in one such case the first operation having taken place as long ago as 1901. Accordingly the value of the post-operative history varies for each individual case. Such general conclusions as are possible will be given and discussed, the tumours being considered under the region involved.

Parotid.—Of the 38 parotid tumours, 6 were recurrent cases when first treated at the hospital, and 5 recurred following a primary operation at the hospital, making a total of 11 recurrent cases in all. Twenty have never developed a recurrence, and are at present (July, 1929) either alive and well, or have died of some other cause without recurrence. Seven cases cannot be traced.

Of the 20 cases free from recurrence, in 2 the time period which has elapsed since operation is nine years, in 4 eight years, in 1 case seven years, in 3 cases six years, in 2 five years, in 2 four years, in 1 case three years, in 3 cases two years, and in 2 one year.

Of the 11 recurrent cases, 4 developed their recurrences within a few months to a year after operation, 3 cases within five years, 3 within ten years, and 1 case as long as nineteen years after operation.

MIXED TUMOURS OF SALIVARY GLANDS 251

Submaxillary.—Of the 6 submaxillary cases, 4 are known to be free from recurrence for varying periods up to seven years; 1 developed a recurrence with which he died within a year of operation, and 1 cannot be traced.

Palate.—Of the 5 palatal cases, 4 are free from recurrence, and 1 developed a local recurrence two years after operation, which is at present being investigated.

Other Regions.—The tongue case cannot be traced; the lip, angle of mouth, and face cases are alive and well, while the tumour of the sublingual was a recurrent case when first treated at the hospital, but is now alive and well.

Summarizing the question of recurrence incidence, we may say that, though a large number of cases treated by operation can survive for long periods without developing a recurrence, yet on the whole the recurrence-rate is relatively high; and since there is one case in which recurrence developed as long as nineteen years after the primary operation, there is no arbitrary time-limit that can be fixed beyond which it can be said that the liability to recurrence ceases.

Influence of Region Involved on Recurrences.—On general principles one would expect that the parotid would be more liable to recurrence than other sites, owing to the fact that operative procedures there are often restricted from fear of damaging the facial nerve, whereas in the submaxillary, palatal, and other regions there is no such anatomical deterrent. An examination of the literature reveals a certain amount of confirmation of this view. The evidence of the present study also points in the same direction, though the number of extraparotid tumours is not large enough to enable any definite conclusions to be formed. The fact, however, that recurrences other than parotid do develop proves that other factors besides ease of surgical removal must be considered in giving a prognosis. This point will be left until the next section for further elaboration.

Clinical Types of Recurrence.—If a recurrence develops, it may take one or two distinct forms. In the first place, an encapsulated tumour may be found resembling in all its clinical features the primary tumour, with the exception that surgical removal may perhaps be slightly more difficult owing to the fibrosis resulting from the previous operation; this may be termed the 'encapsulated type of recurrence'. The other form is the 'infiltrative type of recurrence'; in this type unencapsulated tumour masses may be met with infiltrating locally, spreading upwards to the temple, inwards to the mouth and behind the jaw, and downwards through the tissues of the neck. In addition, fungation through the skin may take place, but metastasis by lymph- or blood-stream does not ordinarily occur. Occasionally a mixed tumour may be incompletely encapsulated from the beginning, and it is in this type of case that cells are especially likely to be left behind at the primary operation. A typical example of local infiltrative recurrences is furnished by *Case 43* of this series, the photographs of which patient are given in *Figs. 157, 158*. *Fig. 159* is an illustration of a non-recurrent encapsulated case for comparison.

Case 43 was a woman who in August, 1908, when 40 years of age, first had a mixed tumour enucleated from the left parotid region. A recurrence developed, which was removed surgically in 1911, and a further recurrence was removed in 1912. In 1917 an infiltrating mass was present in the left parotid region associated with complete facial paralysis on that side, the latter probably being due to operative trauma. The burial of radium resulted in a shrivelling up of this recurrence. In 1926 further buried radium was given for an infiltrating mass projecting both



FIG. 157.—*Case 43.* Clinical photograph of an infiltrative type of recurrence. Since 1917 this patient's recurrences have been kept in check by repeated insertions of radium. No metastases have developed, and the histological appearances are still those of a typical mixed tumour. (The facial paralysis is probably traumatic.)



FIG. 158.—Same case as Fig. 157.

externally and into the mouth, where it was ulcerated at one point. In March, 1929, a recurrent mass was removed from the left submaxillary region, and in July, 1929, radium was inserted into a temporal recurrence. When seen a few months later she was apparently free from signs of active growth. Portions of the tumour removed microscopically in 1927 and 1929 both showed the typical structure of a mixed parotid tumour. At no time were distant metastases found.



FIG. 159.—Case 47. Appearance of an ordinary primary mixed tumour of the parotid.

III. THE CORRELATION OF THE CLINICAL AND HISTOLOGICAL FINDINGS.

It will be remembered that on a histological basis the mixed tumours were divided into four groups: (1) Typical tumours; (2) Cellular, and not well differentiated, but cells similar in type to those of a typical tumour; (3) Cellular, but well differentiated; (4) Cellular, and not well differentiated, and cells showing irregularity and nuclear changes. We will now consider the clinical results in the different histological groups.

1. *Typical Tumours*.—Twenty-nine cases of the series fell into this group. Of these, 18 were free from recurrence, 4 were recurrent cases, and 7 could not be traced. These figures will serve as a standard with which the other groups may be compared.

2. *Cellular and Not Well Differentiated, but Cells Similar in Type to those of a Typical Tumour*.—Nine cases were classified as belonging to this group, of which 8 were free from recurrence at the time of investigation, and 1 was

a recurrent case. This gives a recurrence-rate rather less than that of the typical tumours, though this is a point on which stress cannot be laid owing to the smallness of the figures. It does, however, prove that a high degree of cellularity is no indication of an especial liability to recurrence, provided that the cells maintain the same characteristics as those of a typical tumour. This is an important conclusion since the contrary has often been stated; apart from other considerations, it prohibits this group from being included in the 'cellular and sarcomatous type' of some classifications, as this term by itself implies a special liability to recurrence.

3. *Cellular, but Well Differentiated*.—Six cases fell into this group, of which 1 is free from recurrence, 3 are recurrent cases, and 2 cannot be traced, so that of the 4 cases in which the result is known, 3 have recurred. It must be admitted that the group is so small that any accurate statistical conclusions are impossible; but even so, when one considers that on theoretical grounds it would be expected that a high degree of differentiation would indicate, if anything, a lesser liability to recurrence, the recurrence-rate must be regarded as surprisingly high. Further discussion of this question will be reserved for a subsequent paragraph.

4. *Cellular, and Not Well Differentiated, and Cells Showing Irregularity and Nuclear Changes*.—Eight cases were in this group, of which 2 are free from recurrence, 5 are recurrent cases, and 1 cannot be traced. From this one may conclude that tumours of a high degree of cellularity, with the histological signs of rapid irregular growth, show a special liability to recurrence.

So far, then, our investigations have shown that recurrence may occur in all histological types of mixed tumour, that a high degree of cellularity of itself does not indicate a special liability to recurrence, but that certain types of highly cellular tumour seem to be associated with a tendency to recurrence.

The Causes of Recurrence.—Leaving aside the question of blood and lymph metastasis, which has not occurred in the tumours of the present series, the term 'recurrence' is used clinically to denote the fact that somewhere in the region of the primary operation a second somewhat similar type of tumour has developed. From the strictly pathological standpoint the term would imply that a cell or cells of the original tumour were left behind at the first operation, and that after a greater or lesser interval of dormancy they had taken on active growth, and developed into a second tumour. While this pathological process probably accounts for the majority of cases, it is worthy of note that, as pointed out by Ehrlich, there are two other important causes of clinical recurrence of the mixed tumours, examples of both of which may be found in the present series.

In the first place, the tumours may be multiple at the time of the primary operation, one being large and obvious, and the other a small impalpable or microscopic seedling. If the large tumour only is removed, the seedling tumour remaining may continue to increase in size, and when sufficiently large and superficial constitute what is clinically a 'recurrence', but pathologically merely the normal continued development of a second tumour. It is of course impossible to determine in what percentage of 'recurrences' this

takes place, but one is able to infer the existence of the phenomenon from the fact that occasionally a tumour is removed, and attached to it is found a small seedling nodule, which constitutes quite a separate entity the existence of which was previously unsuspected. Two such cases were met with in the present study. The first is illustrated in *Fig. 160*, which shows the edge of a small spherical tumour, completely encapsulated and separated by loose connective tissue from the large tumour with which it happened to be removed. The second example is seen in *Fig. 161*. Here the main tumour is completely encapsulated, and in the capsule in one instance, and attached to the outer side in the other two, are three quite separate seedling tumours.

The other cause of clinical recurrence is merely the development at a later date in the same gland of another tumour of similar nature. In other words, the 'recurrence' represents



FIG. 160.—Portion of a typical encapsulated mixed tumour which was removed as a small seedling nodule attached to a larger tumour. ($\times 40$.)



FIG. 161.—To show mixed tumour from the parotid, with three seedling nodules in, and attached to, the outer aspect of the capsule of the main tumour. Hemorrhage has occurred into the latter.

the tendency of the salivary gland to develop tumours. For example, in the type of case illustrated by *Case 6* of this series it is difficult to believe that an easily enucleable 'recurrence' which did not appear until ten years after the first operation should be due to cells left behind on that occasion, when the primary operation was also a simple enucleation. But in one case of the series it is possible to affirm quite positively that the 'recurrence' was not due to residual cells of the original tumour, because it developed on the opposite side. This is *Case 14*, which is that of a woman, age 31, who, two years after the enucleation of a tumour of the right parotid region, developed a 'recurrence' not only on the side of the operation, but also on the opposite previously unaffected side. Two and a half years after the second operation she was well and apparently free from growth. The microscopical appearances of the primary and both 'recurrent' tumours were identical.

The present series therefore affords evidence pointing to the existence of two types of clinical recurrence which are not examples of true pathological recurrence of the original tumour: (1) Recurrence due to the continued development of unsuspected seedling nodules, present but not detected at the primary operation; (2) Recurrence due to the appearance of a fresh tumour in a gland which shows a tendency to tumour development. By an appreciation of these facts, the position of at any rate some of the salivary-gland tumours becomes comparable with that of the adenomata of the thyroid gland, in which similar causes of clinical recurrence operate.

It will be convenient at this point to refer to the fact previously noted in this paper that, judged by the small number of cases available for study in the present series, highly differentiated tumours recur with surprising

frequency. In the case last quoted, in which the tendency to tumour formation in the salivary glands was so marked that 'recurrence' occurred bilaterally, a very high degree of differentiation into ducts and alveoli was shown by all the tumours (*see Fig. 149*). This case suggests the possibility that the explanation of the frequency of recurrence of the highly differentiated tumours lies in the fact that glands which have the tendency to the repeated development of tumour formations show also a special liability to tumours of this type. In other words, a high degree of differentiation and the tendency to multiple tumour formation run parallel, and thus the apparent recurrences are not true pathological recurrences at all. This suggestion will obviously require further confirmation before it can be accepted as a fact, but it seems worthy of consideration.



FIG. 162.—Section of an unencapsulated recurrence of a mixed tumour infiltrating beneath the skin. ($\times 40$.)

Type of Recurrence and its Bearing on the Clinical Course.—It has been noted earlier that recurrences may be divided into two categories clinically, the 'encapsulated' and the 'infiltrative'. Histologically also the two types are quite distinct, the former showing the presence of a fibrous capsule as in the original tumour, while in the latter cell masses are found infiltrating the surrounding tissues (*Fig. 162*).

In the present series, of the 11 cases of recurrence in which sufficient information is available to enable a classification to be made, 4 were of the 'encapsulated' and 7 of the 'infiltrative' type. The prognosis of the two types is very different. The encapsulated recurrence may be enucleated in similar fashion to the original tumour, and the prognosis following its removal appears to be no worse than that of a primary operation. Thus *Case 6* remains

quite well nine years after an operation on a recurrence of this type. The infiltrating tumour, on the other hand, is much more difficult to eradicate and the prognosis is correspondingly worse. Numerous and repeated operations are often required, as in *Case 32* of the present series, in which five operations were performed between the years 1912, when the original tumour of the left parotid was removed, and 1928, the final operation being a wide excision of a locally infiltrating mass of growth; microscopical sections of the latter still showed the structure of a simple parotid tumour. It is in this type of case that radium is sometimes so valuable (*see Case 43* already quoted, p. 251).

The Relation between the Microscopical Appearances of the Primary Tumour and the Recurrent Tumours.—The statement is sometimes made that successive recurrences present more and more irregular histological features, until eventually a frank carcinoma results. The material of the present investigation was therefore examined from this point of view. Of the total 14 recurrent cases, in eight no information is available bearing on the question. In two cases the recurrence was similar in microscopical appearance to the primary tumour. In one case, though the section of the primary tumour was not available, a recurrence removed in 1929 was identical with one removed in 1927. In the remaining two cases the sections of the primary tumours were not available, but the reports stated that they were typical mixed tumours: this was the group into which the recurrences also were independently placed after examination. As far as the present investigation goes, therefore, there is no evidence of change of histological type in recurrences.

TREATMENT.

Such observations on treatment as were suggested by the present study may perhaps be briefly given. To deal first with the primary tumours, it is obvious that in situations such as the submaxillary, where a wide excision is possible, such a procedure should be associated with a lesser liability to recurrence than a simple enucleation. In the parotid, however, where the majority of these tumours occur, enucleation must continue to be the surgical method most often indicated, owing to the danger in more radical measures of severing the facial nerve and other important structures. In order to kill any cells inadvertently left behind or any seedling nodules present, the insertion of radium either into or surrounding the tumour cavity seems a wise and rational procedure. This was done in some cases of the present series, but not sufficiently often to enable a statistical evaluation to be made.

The question arises whether radium should replace surgical removal in certain of these cases. There is no information bearing on this question in the present investigation. In the literature, Sistrunk¹⁰ has stated that the experience of the Mayo Clinic has been that radium is not as satisfactory as surgery in this class of tumour. On the other hand, Bland-Sutton has noted that radium sometimes exerts a beneficial influence on the mixed tumours, while the manner in which some of the recurrent tumours of the present series have responded to radium has been very striking. Quick¹¹ has pointed out that all types of mixed tumour do not respond equally well to radium, the more cellular varieties giving the best results, while the acellular myxomatous

growths are rather resistant. For this reason he recommends a preliminary biopsy. At present, therefore, a combination of surgery and radium would appear to offer the best prospect of maximum benefit to the patient, in that it provides the advantages of both forms of treatment.

As for the treatment of recurrences, in the encapsulated type measures similar to those employed for a primary tumour are indicated. In the infiltrative type, while surgical excision is sometimes possible, in view of the extensive procedures often necessary and the resulting deformity, radium therapy would seem to be preferable. In addition, radium is able to afford benefit to cases outside the scope of surgery.

CONCLUSION.

As a result of the present study the conclusion has been reached that the mixed tumours of the salivary glands are a composite group of epithelial tumours, the varying pathological features of which depend on the degree of differentiation attained, the rapidity of division of the cells, and the amount of myxomatous change undergone by the epithelium. According to the degree to which these changes have taken place, all gradations may be encountered between a myxoma on the one hand and a highly differentiated tumour with ducts and acini on the other, and between a slowly growing tumour with regular cells and an irregular anaplastic growth. Based on these factors, a practical pathological classification has been evolved. The combined pathological and clinical study has shown that this classification allows of a certain amount of correlation between the clinical and pathological findings, but that the correlation is by no means complete.

This investigation was carried on while working in the Bland-Sutton Institute of Pathology, Middlesex Hospital, under a grant from the British Empire Cancer Campaign, and is part of an Essay which was awarded the Jacksonian Prize of the Royal College of Surgeons of England for the year 1929. I wish to thank Professor James McIntosh, Director of the Bland-Sutton Institute of Pathology, for allowing me to use the material and the records of the Institute, and for his constant kind interest and help, the Surgeons of the Middlesex Hospital for permission to use their cases, and the Council of the Royal College of Surgeons of England for permission to publish the work.

REFERENCES.

- ¹ WOOD, F. C., *Ann. of Surg.*, 1904, xxxix, 57.
- ² WILSON, L. B., and WILLIS, B. C., *Amer. Jour. Med. Sci.*, 1912, cxliii, 656.
- ³ MCFARLAND, J., *Ibid.*, 1926, clxxii, 804.
- ⁴ BLAND-SUTTON, Sir J., *Tumours, Innocent and Malignant*, 7th ed., 1922, 414. London: Cassell & Co.
- ⁵ KROMPECHER, E., *Ziegler Beitr. pathol. Anat.*, 1900, xxxviii, 1.
- ⁶ EHRLICH, E., *Beitr. z. klin. Chir.*, 1906, li, 368.
- ⁷ FRASER, A., *Surg. Gynecol. and Obst.*, 1918, xxvii, 19.
- ⁸ FRY, R. M., *Brit. Jour. Surg.*, 1927, xv, 291.
- ⁹ EWING, J., *Neoplastic Diseases*, 3rd ed., 1928, 768. Philadelphia: W. B. Saunders Co.
- ¹⁰ SISTRUNK, W. E., *Surg. Clin. N. America*, 1921, Oct., 1515.
- ¹¹ QUICK, D., *Radium Report of the Memorial Hospital*, 2nd ser., 1923, 93. New York: Paul B. Hoeber Inc.

STUDIES IN EXPERIMENTAL MITRAL OBSTRUCTION IN RELATION TO THE SURGICAL TREATMENT OF MITRAL STENOSIS.*

BY W. C. WILSON, EDINBURGH.

TWENTY-EIGHT years ago Sir Lauder Brunton,⁷ impressed by the hopelessness of finding a remedy in medicine for the condition of stenosis of the mitral valve, ventured to suggest that relief might some day be available in this distressing malady from surgical measures. Brunton's proposal, while naturally provoking considerable opposition and adverse criticism, stimulated such interest and effort in the field of cardiac surgery that the procedure he suggested was eventually carried out some twenty years later in operations on human beings. During the past eight years considerable energy, study, and care have been devoted to the development of the operative treatment of mitral stenosis, more especially by Cutler and his co-workers in America. When the situation at the present time is reviewed, it may be said that the position of the physician confronted with this form of cardiac disease is but little more advantageous than it was in Brunton's day. Unfortunately, however, surgery cannot yet offer sure and certain alleviation, and the problem of operative relief is still one of great difficulty. In this paper some aspects of the problem are considered, and an experimental investigation is recorded which, though incomplete, may, it is hoped, be of interest to other workers in this new surgical field.

It is not intended to enter here into a full discussion of mitral stenosis, or to review the history of the development of its operative treatment. A full account of the subject is given in the publications of Cutler and his associates,^{4, 5, 9-14} to which reference should be made. We shall be content to consider shortly the operations already performed, their results, and the grounds upon which they are based.

Operation for mitral stenosis has been carried out on 10 patients: by Cutler in 7 cases,^{9, 13, 14} and by Allen,² Souttar,²⁸ and Pribram²⁶ in one case each. Cutler's first case lived for four and a half years. Souttar's patient is still alive five years after operation, and the remainder died. In one case² the operation was not completed, and in the others death occurred at short intervals after operation. The results of operation are scarcely encouraging. A common experience in all pioneer work in surgery has been initial disappointment, and this new branch has proved no exception.

On what grounds is the operative treatment based? The aim in all cases has been to widen the constricted orifice of the mitral valve and so

* From the Department of Experimental Surgery, University of Edinburgh, under charge of Professor D. P. D. Wilkie.

allow a greater volume of blood to pass from left auricle to left ventricle. The condition of stenosis is thus converted into one of incompetence. There is, of course, no question of restoring the normal condition of the valve. Clinical study gives strong support to the view, which is widely held, that mitral incompetence is a less serious valvular lesion than mitral stenosis. At the present time there seems almost entire unanimity among cardiologists that myocardial damage plays the primary rôle and valvular lesions only a secondary part in cardiac disease. There can, however, be little doubt that in some cases the mechanical obstruction to the circulation in severe mitral stenosis must impose a serious burden on a myocardium already damaged. Advocates of operation argue further that benefit will follow if the valvular lesion of stenosis is changed to incompetence, in which the mechanical embarrassment is not so great. Experimental evidence,^{1, 9, 17} which suggests that stenosis at the mitral valve causes more serious interference with cardiac function than incompetence, has also been cited in favour of operation. The experiments, however, were of the 'acute' type, and the evidence does not seem admissible.

We have conclusive proof that incompetence of the mitral valve can be produced in healthy animals with safety,^{1, 9} and the pioneer work on human cases has demonstrated beyond any doubt that the diseased heart can withstand operative trauma. There is, however, no proof that an operation which changes a long-standing mitral stenosis suddenly to mitral incompetence can readily be tolerated, nor that the change will be beneficial. Such proof is lacking because it has not yet been possible to produce mitral stenosis in animals. Even if the proof were forthcoming, there is still little to guide the surgeon in determining what degree of incompetence he should create. Therefore the primary aim of this investigation was to produce in animals an obstructive condition at the mitral valve resembling mitral stenosis in human beings, to study the effects of the obstruction on the circulation, and later to produce, and observe the results of, a change to incompetence.

EXPERIMENTAL MITRAL OBSTRUCTION.

Attempts to create mitral stenosis in animals have met with little success. Endocarditis produced by micro-organisms usually gave rise to incompetence of the valves.²⁷ Mechanical narrowing of the auriculo-ventricular ring and excision of portions of the left ventricle caused no permanent obstruction, and the introduction of radium around the valve to produce fibrosis was not attended with any degree of certainty.⁹ The most valuable work reported in this field is that of Powers,²⁵ who injected streptococci into the bloodstream after he had damaged the mitral valve by electrocoagulation. Vegetative endocarditis resulted, and was followed some months later by stenosis of the orifice and thickening of the valve leaflets. The mortality from operation and from subsequent endocarditis was considerable. We have not had the opportunity of employing this method.

Production of Obstruction.—To produce obstruction at the mitral valve we first tested methods previously employed by others.^{6, 8} The methods proved difficult and uncertain in results. It was then decided to create an

obstruction to the circulation by inserting and fixing in the heart cavity, across the left auriculo-ventricular ring, a diaphragm of tissue which contained a small central opening. The tissue selected was parietal pericardium, which has a serous covering on both inner and outer aspects. The operation was performed successfully on several occasions, and it was demonstrated that pieces of pericardium could be inserted into the heart cavity without causing clotting. As a method of producing mitral obstruction it possessed several disadvantages. The procedure proved somewhat difficult and time-consuming, and it involved the sacrifice of a considerable portion of auricular appendage, through which we hoped to introduce an instrument in a subsequent operation.

The method of producing mitral obstruction finally adopted will now be described in detail. The greater part of the anterior and left portion of the parietal pericardium was removed and cut into two or more strips of a length greater than the antero-posterior diameter of the left ventricle at the level of the auriculo-ventricular junction. The strips were rolled up to form long solid cords, and to each end of the strip a suture of linen thread was attached. One suture was threaded on a straight round-bodied needle, and the needle and suture were passed through the ventricle from behind forwards. To do this the heart was rotated gently to the right by a silk suture inserted near the apex, and the needle entered at a point about 0.5 to 1 cm. to the left of the posterior interventricular sulcus and immediately below the level of the auriculo-ventricular sulcus, and was made to emerge anteriorly, at the same level, in the angle between the anterior longitudinal sulcus and the auriculo-ventricular sulcus. By pulling on the suture the pericardial strip was drawn across the heart cavity until its anterior end projected through the anterior wall of the ventricle. The ends of the strip remained outside the heart wall and effectively plugged the openings. A second, and if necessary a third, strip was inserted across the cavity on the left of the first at intervals of 2 to 3 mm. The strips were kept gently on the stretch while the ends were stitched to the heart wall. Care was taken in inserting the strips to avoid injury to, or pressure on, the main coronary vessels, and in fixing the strips to avoid any constriction of vessels or heart muscle.

When the operation was successfully performed the strips of pericardium lay across the mitral orifice, thereby narrowing it, and, as they passed through one or both cusps, they tended to fix and immobilize them, thus preventing full closure as well as full opening of the valve. In short, they produced a condition of mitral obstruction to which was added a slight degree of mitral incompetence, thus simulating the condition of mitral stenosis usually found in human beings.

Results of Transplanting Tissue into the Heart Cavity. — Fascia lata transplanted into the heart cavity produces massive clotting which causes death in from three to eighteen days, either by blocking the mitral orifice completely or by embolism. In one experiment, tendon with a synovial covering was employed. No clotting occurred. Parietal pericardium was most readily available, and proved entirely suitable to the purpose. Transplanted strips were examined at intervals of from three days to fourteen months later. There was no obvious change in the appearance of the strip, beyond some

patchy red staining, until about two weeks after transplantation; then its surface appeared smoother and more regular and its consistence was definitely tougher and firmer than previously. From then to about four months after transplantation the strip gradually acquired a smooth, rounded, and usually perfectly regular outline, and a tough inelastic consistence. No changes occurred subsequently. Occasionally smooth rounded nodules were found at the point where the strip pierced the valve cusps. These were formed from small collections of fibrin which became organized after the first week, and were found on the cusp as well as on the strip. Smooth nodular thickenings of the cusps were not infrequent at areas where the valve in its movements impinged on the strip.

Microscopic examination revealed that the strip became gradually compressed into a solid cellular cord with a covering of smooth endothelial-like cells. In some places minute quantities of fibrin were deposited which contained red cells in the meshes. The fibrin ultimately became organized and the area surrounded by an endothelial-like lining. A surprising feature was the presence of well-formed blood-vessels which traversed the strip in the direction of its longitudinal axis.

Effects of Experimental Mitral Obstruction.—In order to estimate the effects of mitral obstruction the following tests were employed: (1) Measurement of the volume of blood passing through the lungs in unit time, calculated according to the principle of Fick¹⁸; this is expressed here as the cardiac output per minute; (2) Electrocardiographic records; (3) Measurement of the heart shadow shown by X rays.

1. *Measurement of the Volume of Blood Passing through the Lungs.*—In the first test the oxygen intake per minute and the oxygen content of the mixed venous blood and of the arterial blood were found. The oxygen intake per minute was divided by the difference in oxygen content of the mixed venous blood and arterial blood, and the result gave the volume of blood passing through the lungs in unit time.

In these experiments the animals were anaesthetized by amytal and morphine in order to prevent struggling or excitement, and the experiments were conducted under exactly the same conditions on each occasion. The anaesthetic dose was 0.35 c.c. of the amytal solution and 3 mgrm. of morphine per kilo. of body weight. Depression of respiration or circulation was not marked. A sample of mixed venous blood was obtained by puncture of the right ventricle, and one of arterial blood from the femoral artery. The samples were drawn under paraffin and oxalated. The oxygen content of the blood samples was estimated in a Haldane's blood-gas apparatus.¹⁹ Simultaneously the air expired by the animal was collected in a modified Douglas bag over a period of ten minutes, its volume measured in a wet gas meter, and a sample analysed in a small Haldane's air-analysis apparatus.²⁰ The oxygen taken up by the animal per minute could then be ascertained. All the data necessary for the final calculation were then available.

Table I shows the results of estimation of cardiac output per minute in normal dogs. The experiments were repeated in each animal on three occasions.

EXPERIMENTAL MITRAL OBSTRUCTION 263

2. *Electrocardiographic Records*.—Records vary greatly with change in position, and it is essential to place the animal in exactly the same position at each examination. In these experiments the animal lay on its back in a special box which prevented any movement or tilting.

3. *X-ray Photographs of the Heart Shadow*.—Photographs were taken on each occasion under identical conditions.

RESULTS.—Experimental mitral obstruction produced by pericardial strips was carried out in 12 animals, 1 cat and 11 dogs. The cat was killed four months after the procedure and a considerable degree of obstruction was

Table I.—CARDIAC OUTPUT IN NORMAL DOGS.

DOG NO.	WEIGHT	OXYGEN INTAKE PER MINUTE	A-V OXYGEN DIFFERENCE	CARDIAC OUTPUT PER MINUTE	OUTPUT PER KILO.	NOTES
	Kilo	c.c.	Vols. per cent	c.c.	c.c.	
9	12.0	59.98	4.30	1395	116	
15	8.6	54.67	5.61	974	113	
30	17.0	80.00	4.10	1951	115	
31	20.0	150.00	5.00	3000	150	Whippet
34	23.0	80.61	4.00	2015	88	
35	6.0	41.18	4.20	980	163	Young dog
36	8.2	32.40	3.30	982	120	
37	13.8	76.04	4.00	1901	138	Young dog
38	19.2	62.79	4.10	1531	80	Heavy old dog

Table II.—CHANGES AFTER MITRAL OBSTRUCTION.

DOG NO.	TIME AFTER OBSTRUCTION	CHANGES IN HEART SOUNDS	ELECTROCARDIOGRAPHIC CHANGES	X-RAY	REDUCTION IN CARDIAC OUTPUT
					Per cent
30	2 months	Roughened first sound	Ventricular extrasystoles. Inversion of T in all Leads	No change	32
31	3 months	Faint mitral systolic murmur	No change	No change	22
35	5 weeks	Faint mitral presystolic and systolic murmurs	Slight reduplication of P wave	No change	25
36	2 months	Roughened first sound	Inversion of T in all Leads (previously in Leads II and III) ..	No change	24

Table III.—CARDIAC OUTPUT BEFORE AND AFTER MITRAL OBSTRUCTION.

DOG NO.	TIME AFTER OBSTRUCTION	OXYGEN INTAKE PER MINUTE		ARTERIAL OXYGEN				MIXED VENOUS OXYGEN				A-V OXYGEN DIFFERENCE IN VOLS. PER CENT		CARDIAC OUTPUT PER MINUTE		
				Volumes per Cent		Saturation per Cent		Volumes per Cent		Saturation per Cent						
		Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After	
		c.c.	c.c.												c.c.	c.c.
30	2 mths.	80.00	70.00	15.0	14.2	93	90	10.9	9.2	68	58	4.1	5.0	1951	1400	
31	3 mths.	150.00	142.00	15.5	15.0	94	90	10.5	8.9	64	53	5.0	6.1	3000	2328	
35	5 wks.	41.18	41.64	14.2	9.9	90	87	10.0	4.2	64	35	4.2	5.7	980	730	
36	2 mths.	32.40	33.75	12.0	12.0	92	88	8.7	7.5	67	58	3.3	4.5	982	750	

found (*Fig. 163*). In the first three dogs of the series the tests revealed no deviation from the normal. The animals remained in good condition. A roughening of the first heart sound was noted in two, while in the third a faint presystolic and a systolic murmur were audible over the apical region. The animals were killed at intervals of from seven to ten months after operation. In the first two the obstruction was slight in degree and probably did not interfere with the function of the heart. In the third a definite obstruction was present (*Fig. 164*) and a slight degree of hypertrophy of the right ventricle was noted.

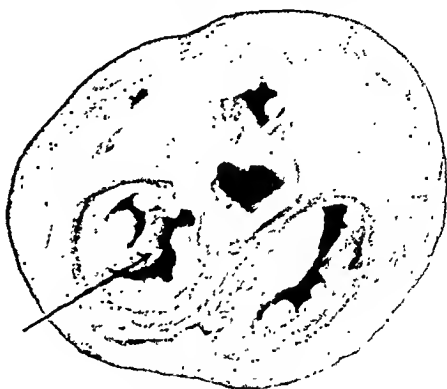


FIG. 163.—Cat. Four months after operation.

FIG. 164.—Dog 17. Seven months after operation.

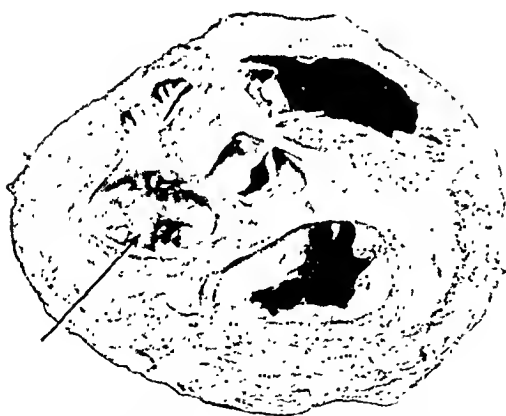


FIG. 165.—Dog 34. Two months after operation.

FIG. 166.—Dog 37. Two months after operation.

FIGS. 163-166.—EXPERIMENTAL MITRAL OBSTRUCTION.
(Arrows indicate obstructing pericardial strips.)

A more marked obstruction was created in the remaining eight dogs of the series. One died twenty-four hours after operation, apparently from the severity of the obstruction, and one died six days later from hæmorrhage due to an error in technique. The remainder recovered. Unfortunately two died suddenly during cardiac puncture two months after obstruction. In both a well-marked obstruction was present (*Figs. 165, 166*). The changes after obstruction in the remaining four animals are shown in *Table II*, and

details of the estimation of cardiac output before and after obstruction in *Table III*. As control experiments tests were carried out at intervals after an operation for removing a portion of pericardium. No definite deviation from the normal was demonstrated by the tests.

Examination of the results shows that the cardiac output was reduced in the four animals by 32, 22, 25, and 24 per cent respectively. The oxygen saturation of the arterial blood did not fall appreciably. Dautrebande, Fetter, and Meakins¹⁵ found that there was a definite reduction in the cardiac minute volume under resting conditions in cases of mitral stenosis with even mild dyspnoea on exertion. They also found that the oxygen saturation of the arterial blood remained within normal limits when no pulmonary complications were present. In these respects, therefore, the experimental findings conformed to those in human cases of mitral stenosis. The oxygen intake remained unaltered after mitral obstruction, the oxygen content of the mixed venous blood fell, and the arterial-venous oxygen difference rose.

Electrocardiographic changes were not constant. In *Dog 30* they were marked. Ventricular extrasystoles and inversion of the T wave in all leads appeared after three weeks and remained. These changes occurring in human beings are usually considered indicative of myocardial impairment. The dog was considerably older than the majority of the animals used. Reduplication of the P wave is a common finding in cases of mitral stenosis. A slight degree of this change was observed in the record of *Dog 35*. In *Dog 36* the electrocardiogram before obstruction showed inversion of the T wave in Leads II and III, and after obstruction in all leads. In no case was there any sign of right ventricular preponderance.

X-ray photographs demonstrated no definite change in size or shape of the heart shadow after obstruction. The operative procedure was tolerated well in almost all cases, and the animals betrayed no obvious signs of heart failure. The response to ordinary exercise was not altered. Examination of the hearts of the animals after death left no doubt that in all cases a definite obstruction had been created at the mitral orifice, which must have impeded the flow of blood from left auricle to left ventricle. Nevertheless no marked dilatation or hypertrophy of any of the heart chambers was present.

The following conclusions can be drawn from the results. A mechanical obstruction was produced to the blood-flow at the mitral valve in all cases. The obstruction was rarely initially formidable, but was progressive to some extent for a period of probably three or four months. The cardiac output under resting conditions was thereby reduced by between 22 and 32 per cent, and its estimation was a valuable test of function. No marked dilatation or hypertrophy of the left auricle or right ventricle resulted, and therefore there were no signs of right ventricular preponderance in the electrocardiogram or change in the X-ray picture of the heart shadow. Other changes in the electrocardiogram were inconstant. Overt signs of heart failure were not observed. It is conceivable that hypertrophy of the right ventricle might have developed had it been possible to give the animals regular strenuous exercise.

RELIEF OF OBSTRUCTION.

For the relief of mitral stenosis two different surgical measures have been advocated. The first, originally proposed by Brunton and later carried out in operations on human beings, consists in widening the stenosed mitral orifice. Jarotsky,²³ of Moscow, suggested another method which, he claimed, would afford relief and would be less difficult of execution than an attack on the valve—namely, to produce an opening in the septum between the auricles. His claim that the formation of an interauricular communication would alleviate the symptoms of mitral stenosis is based on the clinical course of two cases cited by Lutembacher.²⁴ In these cases there was the rare association of mitral stenosis with an interauricular communication; the symptoms of heart failure did not appear until an advanced age and were mild in degree, and life was prolonged until the ages of 74 and 61 years respectively. Both authors explained the unusually favourable course of the cases of mitral stenosis on the assumption that the abnormal communication allowed blood to flow from left auricle to right auricle, and thus the pulmonary circulation was relieved of part of its burden of stasis, which was transmitted to the great veins in the systemic circulation.

It is well, however, to note that in three other cases quoted by Lutembacher where the same anomalies were present, death occurred at the ages of 26, 29, and 41 years respectively. Moreover, there seems some justification for Lutembacher's belief that in all cases the mitral stenosis as well as the abnormal communication was of congenital origin, and adaptive processes were thus in play from the beginning of life. It is difficult to agree with Jarotsky's proposal to form an interauricular fistula for the relief of acquired mitral stenosis. The studies of Holman and Beck^{21, 22} indicate that by this procedure a considerable portion of the blood volume would be short-circuited through the system composed of the auricles, right ventricle, and lungs. To impose this extra burden suddenly on a right ventricle already severely taxed would probably prove far from beneficial. Theoretically there is more justification for producing an interventricular fistula, when the dilated and hypertrophied right ventricle might obtain relief by delivering some of its blood to the left ventricle. The presence of both an interauricular and interventricular fistula would give still more favourable circumstances if the direction of the blood-flow were from left auricle to right auricle and from right ventricle to left ventricle. The volume of blood in the systemic circulation would then be augmented by a mixture of venous and arterial blood. From a practical point of view, however, an attack on the interventricular septum is precluded by the danger of damage to the conducting system of the heart.

Jarotsky's suggestion has been followed in the experimental work of Dmitrieff,¹⁶ conducted mainly in rabbits. In one experiment in our series a fistula was made in the interauricular septum. Mitral obstruction had been created three months before. The opening was made in the septum by a finger invaginating the left auricle appendage, and was large enough to admit the forefinger. The operation was tolerated well. No changes were noted subsequently in the electrocardiogram nor any alteration of the heart shadow.

The animal remained in good condition and was killed seven months later. Examination of the heart revealed a moderate degree of mitral obstruction, and a tiny circular aperture in the interauricular septum with a diameter of about a millimetre. The fistulous opening, probably of a slit-like nature initially, had practically closed. To produce a communication in the septum in a case of mitral stenosis it would be necessary to excise and remove a piece of septum. The experiment yields no evidence in regard to the value of an interauricular communication in mitral stenosis.

Widening the Orifice of the Mitral Valve.—When the procedure of widening the stenosed orifice is considered, the problems are found to be largely technical. Some controversy has arisen concerning especially the route of approach to the valve and the method of relieving the constriction.

Method of Approach.—The valve has been approached from above through the left auricular appendage and from below through the left ventricle. The former approach is favoured by Allen and Graham¹⁻³ on the grounds that serious irregularities and disturbances of heart action follow the introduction of an instrument through the ventricle, while, as they point out, the left auricular appendage can be clamped, cut, sutured, or even excised without any apparent detriment to the function of the heart. This approach was employed by Souttar in his operation. Cutler at one time upheld the approach through the ventricle, which is much more accessible than the auricle, denied the occurrence of frequent serious irregularities from interference with the ventricle, and stressed the ease by which hæmorrhage from the ventricle can be controlled. His experience of operations on mitral stenosis, where he could view the thin-walled, greatly dilated left auricle, convinced him that an approach from above could not be a sound surgical measure. Recently he has modified his views, and now looks with more favour on an approach from above.

It may be noted that the irregularities of heart action observed by Allen and Graham in animals followed attempts to introduce their cardioscope through the ventricle, and to tie it in. It is not surprising that accidents occurred in these circumstances. If Cutler's technique and valvulotome are used, serious irregularities of action are comparatively infrequent. There is no doubt also that in human beings the auricle lies far back in the thorax and is much more inaccessible than the ventricle, and Cutler considers that consequently it is necessary to use a curved instrument when approaching from the auricular appendage.

On the whole, our preference is for the approach through the auricular appendage. In a large number of experiments in this investigation the appendage was clamped, incised, and used for the introduction of some instrument, and no serious accident could be attributed to interference with the appendage. In addition, it seems likely that in operations on mitral stenosis the difficulties of locating the valve will be considerably less than when the approach is through the ventricle. The instrument introduced from above will be guided by the shape of the cavity to the constricted orifice, more especially when the valve is of the funnel-shaped or 'fish-mouth' type. One further possible advantage of approaching from above

is that the instrument can be introduced through a sheath tied into the appendage. To tie a sheath into the ventricle would prove dangerous and difficult.

Relief of Constriction.—Different methods suggested for the relief of the constriction are: (1) Section of the valve; (2) Excision and removal of a piece of valve; and (3) Dilatation of the orifice. There is little to recommend section or simple incision of the constriction. It seems unlikely to confer any great benefit in widening the orifice, especially when the valve is tough, rigid, and possibly cartilaginous, and to cut such a valve with a thin-bladed knife might prove extremely difficult, if not impossible. Souttar dilated the constriction with his finger. The stenosis in his case was not, apparently, of an extreme degree. Treatment by dilatation of pathological stricture in other parts of the body must be intermittent and repeated in order to achieve success, and the same is probably true for stricture of the mitral valve. Repeated instrumental dilatation of the mitral valve does not appear as yet to be feasible. For the moment the most reasonable method seems to be excision and removal from the heart of a piece of the mitral valve. Further information on this point may be forthcoming if mitral stenosis can be produced experimentally by the method of Powers.

Use of Vision.—For consistent success in surgical measures the aid of vision is essential. The invention of a cardioscope by Allen and Graham was therefore a notable achievement. Their original paper should be consulted for the details of the instrument. Cutler tried to combine the principles of the cardioscope in his cardio-valvulotome with its extremely powerful cutting device, but apparently was not impressed with the vision afforded. The following experiments were conducted with a view to establishing a method of operating on cases of mitral stenosis, and it seemed obligatory to employ an instrument by which the valve could be cut under the guidance of the eye.

PERSONAL EXPERIENCES.

To widen the orifice of the mitral valve, and so produce mitral incompetence, the valve was approached from above. Through the left auricular appendage an instrument was introduced by which a piece of valve could be excised and removed, using the principle of operating under vision. For reasons already stated, the method is the most likely to achieve success in operations on human beings for mitral stenosis.

The Instrument.—This is built around a straight hollow metal tube, the ends of which are closed with glass windows. Illumination is provided by a battery in the large proximal handle, which also contains a bulb, a focusing lens, and a switch at its lower end. The light is reflected down the main tube by a reflecting mirror with a central opening.

On the outside of the tube are the parts for partial valvulotomy, which comprises excision of a piece of valve and its removal from the circulation. This section is in two portions—one a ring, for trapping the valve and holding it against the distal window, and the other a circular shear which passes distally outside the ring and cuts the portion of valve enclosed in the ring. The ring has three teeth projecting proximally, and is mounted on a rod

which runs in a groove on the under surface of the instrument and ends proximally in a collar. To the collar are attached a handle on the under surface, and a spring catch which works on a ratchet on the side of the instrument. The collar is held against a circular spring by the catch engaging in the teeth of the ratchet. The circular shear has a sloping cutting edge and is borne on a rod which runs on the upper surface of the instrument and ends in a handle, which works against a spring. The distal portion of the instrument behind the shear and ring is furnished with an extra casing which is perfectly cylindrical in shape and encloses the rods bearing the ring and shear.

An accessory portion of the instrument is the sheath. This is a light metal tube of constant bore. At its middle is a thin metal platform which swings outwards on a hinge. The platform, when engaged, closes the lumen of the sheath, forming a watertight diaphragm, and when swung outwards leaves

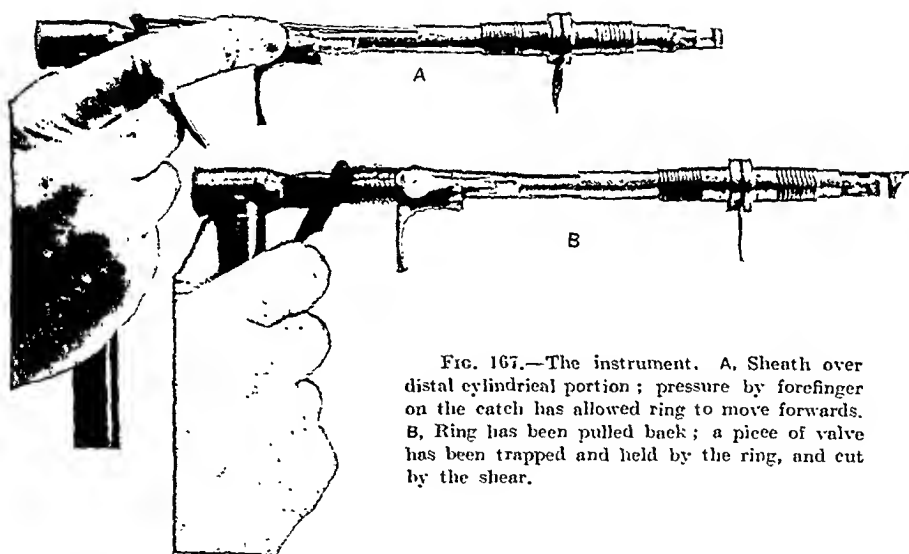


FIG. 167.—The instrument. A, Sheath over distal cylindrical portion; pressure by forefinger on the catch has allowed ring to move forwards. B, Ring has been pulled back; a piece of valve has been trapped and held by the ring, and cut by the shear.

the lumen entirely clear. At the ends of the sheath are a number of grooves which give a firm grip to a ligature. The bore of the sheath is 9.5 mm. The distal cylindrical part of the instrument fits the sheath closely. The instrument can pass smoothly through the sheath without allowing the escape of blood. Its total length is 25 cm.; the outside diameter of the ring is 8 mm. The instrument will cut and remove a piece of valve which is almost circular in shape and measures 8.5 mm. in diameter. It can be sterilized by immersion in carbolic solution or by formalin vapour. Boiling causes condensation on the inside of the windows. The battery is inserted into the main handle after sterilization of the instrument.

Technique of Partial Valvectomy.—The left auricular appendage is clamped at its base by a weak-jawed clamp with curved blades protected by rubber. The appendage is opened by cutting off its tip and its cavity

washed out with normal saline solution. The sheath is then tied into the appendage by a purse-string suture reinforced by a ligature. Waxed silk or linen thread is employed as suture material. The sheath platform is closed and the clamp released. The instrument is introduced into the sheath, the platform opened, and the instrument passed at once into the heart cavity. The light is switched on. As it closes the mitral valve impinges on the end of the instrument, and may be seen at the moment of impact. The operator aims at excising a portion of the aortic cusp of the valve. Pressure on the catch is made by the forefinger, and the ring passes into the ventricle beyond the valve (*Fig. 167 A*). Traction on the ring handle brings back the ring, which then holds a piece of cusp against the distal window. The cusp is held securely by the teeth in the ring, and the position of the ring is fixed by the catch engaging in the teeth of the ratchet. The trapped portion of the valve is in close contact with the window and can be examined by the eye at leisure. The valve is then cut by manipulating the shear handle (*Fig. 167 B*). Repeated strokes of the shear can be made if the valve is very tough. The excised portion of valve is held by the ring and removed from the circulation when the instrument is withdrawn. As the distal end of the instrument passes beyond the middle of the sheath during withdrawal the platform is closed. Finally the base of the appendage is clamped and tied. The stump may be cut away.

Criticism.—In animals with normal healthy valves the operation is not difficult. It can be carried out without the loss of any blood. The use of a sheath is attended with several advantages over the method of tying the end of the instrument into the appendage. Introduction is easier and safer, exploration and examination of the heart cavity can be made with greater facility and over a wider range, the instrument can be withdrawn for the purpose of cleaning it or to examine the excised portion of valve, and can be re-inserted immediately to repeat the procedure if necessary.

On the whole the instrument combines satisfactorily the principles of the cardioseope and the powerful cutting mechanism of the valvulotome. The portion of valve trapped by the ring is held securely by the teeth in the ring, is brought against the distal window, where it can be examined by the eye, and, after being cut free, is removed from the circulation. The shear is an efficient cutting device. A special advantage of the arrangement for excision is that repeated strokes of the shear can be made to cut the valve without any risk of losing the excised piece. With Cutler's valvulotome it is necessary to complete with one stroke the separation of the piece between the cutting edges. The lighting system has two main conveniences: (1) There are no connecting wires, and thus manipulation of the instrument is facilitated; (2) The arrangement of a proximal reflecting mirror gives a larger field for examination than the system in which the light is supplied by a small bulb set inside the tube close to the distal end. The area available for examination through a narrow tube is considerably reduced by the latter system, although this provides superior illumination. The sloping mirror reflecting light from a large bulb gives better illumination than an arrangement of small bulb and prism as used in an ophthalmoseope.

The feature of the instrument and method which is least satisfactory

is the visual section. The degree of vision is disappointing, and certainly does not permit of easy and rapid identification of structures. Cutler also, in his attempt to design a cardioscopic valvulotome, had a similar experience. An invention providing good vision in opaque media such as blood is probably essential to the solution of this problem.

Experiments in Partial Valvulectomy.—

1. *On Normal Animals.*—A piece of mitral valve was excised in three dogs. The operations were tolerated well, and the animals were killed at intervals of from six to fourteen months later. In all there was some degree of hypertrophy of the left ventricle. The edges of the artificial valvular defect were smooth and rounded. There was no sign of clotting of blood.

2. *On Animals with Mitral Obstruction.*—Three dogs (Nos. 30, 31, and 36) were subjected to operation. Mitral obstruction had been present for six months, four months, and three months respectively, and the results of obstruction in each have already been quoted in *Tables II* and *III*.

Dog 30 died during the operation. There was definite evidence of myocardial impairment before the procedure. A marked degree of mitral obstruction had been created. At the operation an obstructing pericardial strip with an attached piece of mitral valve cusp was first excised. To ensure a definite incompetence of the valve a further piece of the aortic cusp was removed. Shortly afterwards the heart dilated and stopped beating. Death was due, apparently, to the sudden production of an excessive degree of mitral incompetence.

Dog 31 died two days after operation. Examination showed œdema of the lungs. The heart seemed unable to tolerate the sudden change from obstruction to a fairly well-marked incompetence of the mitral valve.

Dog 36 made an excellent immediate post-operative recovery. The animal was found dead on the morning of the sixth day after operation. Death was unexpected, and no obvious abnormality was found post mortem. The excised portion of valve was smaller than in the other animals.

It would not be permissible to draw conclusions from these experiments. The number of experiments is too small, and the animals have not survived for a period sufficient to afford an opportunity of estimating the effect of the operation by physiological tests. There are, however, indications that the heart cannot easily withstand a sudden change from obstruction at the mitral valve to a marked degree of incompetence. It has been shown by Cutler that the healthy animal will succumb if a severe grade of mitral incompetence is produced. During this investigation experience of the operation of partial valvulectomy in normal animals and in animals with mitral obstruction suggests that in the former a larger piece of mitral valve can be removed with safety than in the latter.

TECHNICAL CONSIDERATIONS OF EXPERIMENTAL CARDIAC SURGERY.

Success in an investigation such as has been described depends largely on surgical technique, and consequently the very best conditions for operative work are required. Fortunately these were available in the Department of Experimental Surgery at Edinburgh. The most important requirements are

a safe and reliable method and control of anæsthesia, a rigorous aseptic régime, and correct management of operative technique.

The majority of the experiments were carried out on dogs, a few only on cats. In both the heart is approached by the transpleural route, therefore some form of positive pressure apparatus is required for anæsthesia. We employed intratracheal insufflation of ether. A current of air from an air-pressure pump is led through a bottle of ether; provision is made for mixing ether vapour and air and also for alternating the current. A gum-elastic or straight metal tube is introduced into the trachea and connected with the pressure apparatus. If a gum-elastic tube is employed, it should be protected in the mouth by a gag. The tube should so fit the opening of the glottis that air can return freely while the lungs are kept distended by a current of air under moderate pressure during the time the thorax is open. If the intratracheal tube is too large, regulation of the concentration of anæsthetic vapour in the lungs becomes very uncertain, while with a tube too small it is difficult to secure efficient aeration without excessive ventilation of the lungs and consequent loss of carbon dioxide.

The standard of asepsis must not be lower than that for operations on human beings. The operations in these experiments may be of considerable duration—even as long as three hours—and it is all-important to avoid infection from the skin surface by excluding the skin rigidly from the field of operation.

The thorax is opened through the fourth left intercostal space. Excellent access to the heart is afforded by this incision. To minimize adhesions after operation the lungs are protected by moist wool, and all cleansing in the thoracic cavity is done by suction over pieces of wool soaked in normal saline solution.

A few general principles of operations on the heart itself may now be enunciated. Control of the position of the heart is most easily maintained by using a silk suture inserted near the apex, as described by Cutler. Care is necessary not to kink the pedicle of the heart during manipulation. Damage to the main coronary vessels and to the conducting system must be avoided. Fine silk is the best material for suture or ligature. Cutler's method of crossed sutures efficiently controls hæmorrhage from the ventricle. Hæmorrhage from the auricles should be controlled by the use of a weak-jawed clamp and by subsequent ligature in order to approximate endothelial surfaces on the internal aspect and so prevent intracardiac clotting. For suture of the ventricles fine curved round-bodied needles are employed and a minimum of muscular tissue is included by the suture.

Several complications may arise during operation. When the heart is first exposed the auricles not infrequently pass into a state of flutter. The ventricles are unable to respond to the high auricular rate, and heart-block of 3 to 1 or 2 to 1 occurs. Though usually but momentary, the irregularity may be more persistent or may recur several times. The disturbance seems to be provoked by the mechanical stimulus of an instrument touching the left auricular appendage. Excitability to mechanical stimuli gradually lessens during operation. The heart may stop, following damage to the coronary supply or conducting system.

Apart from these, the gravest risk is from anoxæmia. When the thoracic cavity is opened the pressure is regulated, allowing a slight degree of collapse of the lungs in order to facilitate exposure of the heart. If the collapse is long-continued, some degree of stasis develops in the pulmonary circulation. Wrinkled blue patches appear over the surface of the lung, and ultimately deficient oxygenation of the arterial blood is witnessed by cyanosis observed in the heart itself. The lungs should be fully inflated at intervals to prevent the condition. Serious anoxæmia results in weakening and slowing of the heart-beat, and finally dilatation of the chambers and stoppage.

The best stimulant to the heart during arrest is manual massage and full inflation of the lungs, especially if there has been cyanosis. The circulation can be continued artificially by massage of the ventricles. Adrenalin injected into the cavity or dropped on the surface of the heart is efficacious also. Hot saline damages the epicardial lining.

Danger after operation may arise from 'heart tamponade'. To avoid this the edges of the opening in the pericardium should be approximated with interrupted silk sutures set at least one centimetre apart. Any reactionary effusion will thus be enabled to pass into the pleural cavity.

During closure of the wound the lungs are kept inflated by continuous pressure to expel air from the pleural cavity. The ribs on either side of the intercostal incision may be brought together by one or two sutures to relieve the tension on the muscles. Accurate approximation of the divided muscles in layers is essential for good healing. No skin dressing is required.

On the experience of more than fifty operations in animals it can be affirmed that the heart is remarkably tolerant of operative trauma. Two animals died during operation—one from failure of the anæsthetic apparatus, and one as a result of operative procedure. Two died within a few days of operation as a result of avoidable errors in technique—one from sepsis and one from hæmorrhage. Five died at short intervals after operation as a result of the operative procedure—two from intracardiac clotting after the insertion of fascia lata to produce mitral obstruction. Death was unavoidable in three of the nine fatalities, and the mortality from operation might be estimated at 12 per cent. In half the fatal cases death followed a second operation of partial valvulotomy for mitral obstruction. The remaining animals lived in good health until killed for examination at intervals up to fifteen months after operation. An operation on the heart is certainly tolerated by the healthy animal at least as well as an operation of the same duration, and of comparable severity, on any abdominal organ.

SUMMARY.

1. Surgical treatment of mitral stenosis involves converting the condition of stenosis into incompetence of the valve.
2. There is as yet no proof that the heart in which the mitral valve is stenosed can readily tolerate a sudden change to incompetence, nor that such a change will be beneficial.
3. In an experimental investigation it was decided to study the effects

of a mechanical obstruction at the mitral valve, and of a subsequent change to incompetence of the valve.

4. An original method was evolved of producing mitral obstruction, and the effects of obstruction on the heart and circulation are recorded.

5. Methods of surgical relief for mitral stenosis are discussed. A new instrument for performing partial valvulotomy under vision is described.

6. Experiments in converting mitral obstruction to incompetence are not of sufficient number and duration to offer evidence of value on the main question. The investigation is not yet complete.

7. Experiences in the technique of operations on the heart in animals are contributed.

I wish to record my appreciation of the continued interest shown by Professor D. P. D. Wilkie and of the encouragement received from him during the investigation. For the electrocardiographic records I am indebted to Mr. J. Y. Bogue, of the Pharmacology Department, University of Edinburgh. The work was carried out during tenure of the British Medical Association Research Scholarship and the McCunn Medical Research Scholarship. The expenses were in part defrayed by a grant from the Moray Fund, University of Edinburgh.

REFERENCES.

- ¹ ALLEN, *Arch. of Surg.*, 1924, viii, 317.
- ² ALLEN, *Arch. franco-belges de Chir.*, 1925, xxviii, 394.
- ³ ALLEN and GRAHAM, *Jour. Amer. Med. Assoc.*, 1922, lxxix, 1028.
- ⁴ BECK, *Ann. of Med. History*, 1926, viii, 224.
- ⁵ BECK and CUTLER, *Jour. of Exp. Med.*, 1924, xl, 375.
- ⁶ BERNHEIM, *Bull. Johns Hopkins Hosp.*, 1909, xx, 107.
- ⁷ BRUNTON, *Lancet*, 1902, i, 352, 547.
- ⁸ CUSHING and BRANCH, *Jour. of Med. Research.*, 1907-8, xii, 471.
- ⁹ CUTLER, LEVINE, and BECK, *Arch. of Surg.*, 1924, ix, 689.
- ¹⁰ CUTLER and LEVINE, *Boston Med. and Surg. Jour.*, 1923, clxxxviii, 1023.
- ¹¹ CUTLER, *Arch. of Surg.*, 1926, xii, 212.
- ¹² CUTLER, *Arch. franco-belges de Chir.*, 1925, xxviii, 376.
- ¹³ CUTLER and BECK, *Nelson's Loose-Leaf Surgery*, 1927, 233. New York: Thos. Nelson & Sons.
- ¹⁴ CUTLER and BECK, *Arch. of Surg.*, 1929, xviii, 403.
- ¹⁵ DUTREBANTE, FETTER, and MEAKINS, *Heart*, 1922, x, 153.
- ¹⁶ DMITRIEFF, *Zentralb. f. Chir.*, 1926, liii, 715.
- ¹⁷ DRINKER, PEABODY, and BLUMGART, *Jour. of Exper. Med.*, 1922, xxxv, 77.
- ¹⁸ FICK, *Verhandl. d. Würzb. physikal-med. Gesellsch.*, 1870, N.F. ii, 16. (Quoted by Stewart and Gilchrist, *Jour. Clin. Invest.*, 1928, v, 335.)
- ¹⁹ HALDANE, *Jour. Pathol. and Bacteriol.*, 1920, xxiii, 443.
- ²⁰ HALDANE, *Methods of Air Analysis*, 1920, 3rd ed. London: Griffin & Co.
- ²¹ HOLMAN, *Arch. of Internal Med.*, 1925, xxxvi, 516.
- ²² HOLMAN and BECK, *Jour. of Exper. Med.*, 1925, xlii, 661.
- ²³ JAROTSKY, *Zentralb. f. Chir.*, 1926, liii, 140.
- ²⁴ LUTENBACHER, *Presse méd.*, 1925, xxxiii, 236.
- ²⁵ POWERS, *Arch. of Surg.*, 1929, xviii, 1945.
- ²⁶ PRIBRAM, *Arch. f. klin. Chir.*, 1926, cxlii, 458.
- ²⁷ ROSENOW, *Jour. Amer. Med. Assoc.*, 1915, lxy, 1687.
- ²⁸ SOUTTAR, *Brit. Med. Jour.*, 1925, ii, 603.

SOME COMPLICATIONS OF HYDATID DISEASE.*

By HAROLD DEW,

PROFESSOR OF SURGERY AT SYDNEY UNIVERSITY.

A FULL consideration of the various complications which may occur during the life of a hydatid cyst is beyond the scope of this article, but it is felt that a general survey is possible, and that in such a manner attention will be drawn to some recent advances in the pathology and clinical aspects of this disease which are not yet sufficiently appreciated and are of fundamental importance to an understanding of much that has been misinterpreted in the past. In all countries it is the onset of complications which alone determines the first clinical manifestation of hydatid disease in nearly three-quarters of the cases, so that, although no new facts will be brought forward in this review, it will require no apology if it succeeds in reviving interest in some of the most important of these complications and their sequelæ.

THE TYPICAL UNCOMPLICATED CYST.

Development.—Following ingestion of the ovum, the hexacanth embryo hatches in the upper part of the alimentary canal, makes its way into radicles of the portal vein, and as a rule lodges in the liver. A hydatid follicle is formed there by the accumulation of mononuclear and eosinophil leucocytes around the parasite, which becomes vesicular at the end of two or three weeks. Fluid is formed within the laminated layer, and ultimately, with the same dramatic changes which characterize the growth of all embryos, a univesicular cyst is formed (*Fig. 168*). As this grows the tissue reaction in the host leads to the formation of an adventitious capsule. At first this is highly cellular, but as the parasite elaborates a semipermeable laminated cuticle, and the absorption of hydatid products ceases, it becomes replaced by concentrically arranged fibroblastic cells. Further extension of the cyst takes place by a quiet pressure necrosis of surrounding tissue and its replacement *pari passu* with fibrous tissue. This adventitia merges gradually and intimately into the tissues of the host, and in old large cysts may become very thick and dense.

Huxley introduced the term 'ectocyst' to denote the fibrous tissue derived from the host, to distinguish it from the parasitic layer, which he called 'endocyst'. These terms have led to endless confusion, and I consider that they should be abandoned—the term 'cyst' being used for the parasite, and 'adventitia' for the fibrous capsule derived from the host.

In its fully developed state the cyst consists of a double-layered parasite with enclosed specific fluid in which float the reproductive elements.

* Based on a Hunterian Lecture delivered at the Royal College of Surgeons of England on July 10, 1930.

The outer layer of the parasite or the laminated membrane is a creamy-white structure which has remarkable properties and important functions. It varies in thickness with age and is laid down by the inner nucleated germinal layer. Chemically it is composed of hyalin—a peculiar substance somewhat allied to chitin. Histologically it is homogeneous, it contains no nucleated material, and is arranged in concentric laminæ, so that it resembles no other pathological product. The laminations are laid down from within out, so that the outer are stretched more than the younger inner layers. As a result they often show fissures and signs of moulting, and when the cyst is torn they contract more than the inner so that the whole cyst tends to turn inside out. The larger the cyst and therefore the thicker the laminated layer, the more complete is this eversion.

It is possible that this peculiarity may have a biological significance in that it ensures shedding of the reproductive elements and makes them readily

accessible to the carnivorous definitive host. This layer also gives rigidity and support to the delicate germinal layer and to the developing reproductive elements. In addition it has a special selective permeability, which not only ensures retention of the specific hydatid fluid at a fairly high tension, but also prevents the entrance of noxious substances from the host. It is therefore above all a protective layer, and on its integrity seems to depend the maintenance of ideal conditions for the development of the all-important reproductive elements.

The inner delicate nucleated germinal layer is in close contact with the laminated layer. It elaborates both the laminated membrane

and the specific fluid, and from it are developed the scolices or future worm heads. The specific fluid is clear, carries only a trace of protein, but has a very highly saline content and contains some specific hydatid substance. It is a nutritive fluid, and no doubt must be kept at a constant composition for normal development of the scolices to proceed.

The scolices, unlike those of cestodes, are developed in groups inside specialized sacs or brood capsules. They are just visible to the naked eye (measuring up to 160μ in the resting state), and are seen in all stages of development—from the undifferentiated cellular bud to the fully developed scolices with suckers and hooklets and contractile tissue. They are in reality the larval form of the worm, and may be present in thousands in a single cyst. With the formation of scolices the cyst has reached its full development, and it is only necessary for some of them to be ingested by the correct



FIG. 168.—Section of the wall of a typical univesicular hydatid cyst, showing laminated layer, blood capsules, and scolices. (Oc. 2; Obj. 3.)

definitive host for the life-cycle to be completed by the development of mature ova-producing worms.

Clinical Aspects of the Simple Cyst.—These simple cysts are typically found in children and young adults. It is extremely important to realize that the majority of hydatid, like all parasitic, infestations take place in childhood, so that the majority of cysts are nearly as old as the patient harbouring them. This is borne out by evidence from all sides, and its recognition has had a profound influence on our appreciation of much of the pathology of the disease.

The outstanding element characteristic of these cysts is their latency. They may exert pressure on any structure, and, since practically any organ may be involved, it can readily be understood that bizarre and protean manifestations may be produced. Small cysts in special situations such as the orbit, the cranial cavity, or the spinal canal may of course produce grave symptoms relatively early. Deformity may be produced, especially in children, but it is notable that large cysts can be tolerated. This is not to be wondered at when one considers the frequency of infestation during the growing period, the extremely slow growth of the cyst which enables compensatory changes to occur, and the fact that cysts are often shut off from the body fluids of the host by a relatively impermeable fibrous adventitia.

Ultimately, however, some complications of the cyst occur, and it is obvious that the frequency of such complications increases as age advances and the cyst increases in size. It is found that they are relatively rare in young subjects, manifesting themselves usually between 25 and 80 years of age. Practically all these complications depend on a previous escape of fluid from the cyst, this varying from a slight leak, which is often unnoticed or masked by other symptoms, to a frank rupture.

Rupture of the Cyst.—As the cyst enlarges it may encroach on a natural channel, a hollow viscus, or serous cavity—one area of the laminated membrane thus becoming relatively unsupported. As a result it may give way spontaneously, even during sleep, following muscular movement, coughing, or straining, or more commonly following direct mild or severe trauma such as a blow, a fall, a crush, or a perforating injury. As a result the tear in the laminated membrane, which rapidly enlarges owing to the peculiar grain of this structure, allows the escape of the contained fluid and hydatid elements. Such rupture takes place most commonly into the subcutaneous or muscular tissues, into the bile-ducts, bronchi, alimentary canal, or urinary tract; into a serous cavity such as the peritoneum, pleura, or pericardium; or into the chambers of the heart or large veins. More rarely rupture occurs into the ventricles of the brain, the spinal theca, or on to the exterior of the body.

Each of these has its own characteristics, and as some of them may be combined in the individual case it will be readily understood that a complete discussion is outside the scope of this review. It is found, however, that the sequelæ may be grouped together in the following way:—

(1) General—applicable to practically all types: (a) *Immediate*—hydatid anaphylaxis; (b) *Delayed*—secondary echinococcosis.

(2) Special—applicable particularly to cases of rupture into a natural channel: (a) *Immediate*—mechanical effects; (b) *Delayed*—suppuration in the cyst.

HYDATID ANAPHYLAXIS.

Many clinicians have noted the appearance of peculiar toxic manifestations following puncture or rupture of hydatid cysts. The most common of these is the hydatid rash, characterized by irregularly distributed urticarial wheals with general or localized erythema and pruritus. Other symptoms of variable occurrence are dyspnoea and cyanosis, abdominal pain, vomiting and diarrhoea, syncope, delirium, or nausea. Profound cardiovascular shock may dominate all the symptoms, and on rare occasions lead to fatal termination.

The close resemblance between the clinical picture in these cases and that associated with anaphylaxis due to foreign proteins, the presence of sensitiveness to hydatid fluid in patients with hydatid disease as shown by the well-known intradermal test of Casoni, together with the variable power of inducing passive sensitization by injecting their serum into suitable animals, make it almost certain that the majority of the symptoms which follow rupture of hydatid cysts are anaphylactic in nature.

A patient with hydatid disease may absorb varying amounts of hydatid antigen. This absorption probably takes place readily during the early stages of development of the parasite before the impermeable laminated membrane is fully elaborated. The occurrence of a specific cellular reaction around the early follicle favours the view that products of parasitic metabolism may then be diffused, and at the same time, since the infection is doubtless often multiple, other embryos may be destroyed in the tissues with the liberation and absorption of hydatid antigen. As a result, specific sensitization of the cells of the host takes place, and if at any later period even small amounts of hydatid fluid are absorbed into the circulation, anaphylactic symptoms of varying severity occur. The degree of sensitization varies greatly, but the relatively high percentage of positive findings with the intradermal test of Casoni shows that it can occur even in uncomplicated cysts in which leakage of fluid has not occurred.

There is no doubt that during the life of most cysts leakage does occur, and that, were more accurate histories possible, clinical evidence of anaphylaxis would often be obtained. In some instances the only effect may be the production of desensitization, but in the majority of cases symptoms would occur. Following the initial symptoms recovery is the rule, but after a period of two or three weeks the patient may become again sensitized, so that a subsequent leakage may be followed by even more striking manifestations. That some of these patients become extremely sensitive is borne out by the fact that I have seen the most alarming symptoms after the intradermal injection of 0.25 c.c. hydatid fluid for the Casoni test. As pointed out above, the clinical picture is very variable, and *Table I* emphasizes this.

Clinically there are three main types of cases—the common benign, the severe, and the grave cases. In the first type the usual manifestations are cutaneous, but there are often other symptoms such as faintness, dyspnoea, vomiting, and irregular pyrexia.

Table I.—ANAPHYLACTIC SYMPTOMS IN 20 CASES OF HYDATID DISEASE.

Site of cyst	Complication	Casoni Test	Dyspnea	Cough	Cyanosis	Cardiac	Agitation	Pvrexia	Urticaria	Pruritus	Swelling	Delirium	Ptychomania	Dilated pupils	Death	Remarks
Liver	Bile-duct rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	Three attacks
Heart	Rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	Repeated post-operative
Lung	Cardiac rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	Many previous operations
Spine	Rupture of D. cysts	?	+	+	+	+	+	+	+	+	+	+	+	+	+	In convalescence
Liver	Bile-duct rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	Very small dose
Liver	Bronchial rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	Post-operative
Liver	Casoni test	?	+	+	+	+	+	+	+	+	+	+	+	+	+	Post-operative
Lung	Rupture into P. artery	?	+	+	+	+	+	+	+	+	+	+	+	+	+	Many previous operations
Liver	Peritoneal rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	After eight days
Liver	Pleural rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	Post-operative symptoms
Liver	Biliary rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Liver	Pleural rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Liver	Bile-duct rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Lung	Pneumothorax	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Liver	Rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Omentum	Leak	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Pelvis	Leak	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Omentum	Leak	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Liver	Post-operative	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Liver	Peritoneal rupture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	
Liver	Puncture	?	+	+	+	+	+	+	+	+	+	+	+	+	+	

D Delayed reaction.

Severe cases are quite common, particularly after exploratory puncture or rupture into a serous cavity. It is important to remember, however, that similar cases may follow rupture into the bronchi, bile-ducts, or any other natural channel. Dyspnoea, cyanosis, collapse, nausea, vomiting, and weak pulse are the common initial symptoms. Urticaria may be entirely absent in the early stages, or be delayed for thirty-six hours, when it may lead to a retrospective diagnosis; occasionally it may not be observed at any stage. The classical examples occur following rupture of a hepatic hydatid into the peritoneum, the condition not infrequently being regarded by the clinician and patient as gastro-enteritis or food poisoning, particularly if there is a history of the ingestion of fish of any description. The dramatic onset with pain, the rapid recovery within a few hours, the intense nature of the urticaria, the non-persistence of vomiting and diarrhoea, and occasionally the previous history, should make one suspect that it is not gastric in origin. It must be remembered, too, that the peritoneum is surprisingly tolerant of the non-irritating hydatid fluid, and after the first few hours abdominal rigidity and tenderness may be completely absent in these cases. All of them react dramatically to an intravenous injection of 10 min. of adrenalin.

The grave cases are rare, occurring typically after rupture into the cardiovascular system, although fatal cases have been recorded after simple puncture of a cyst. Pulmonary and nervous symptoms predominate, urticaria and erythema being rare, although pruritus may be intolerable.

It is necessary to mention another type of anaphylactic phenomenon, and we are indebted to Dévé¹ for drawing attention to it. As general anaesthesia abolishes the anaphylactic state, it is rare for grave anaphylaxis to occur during or just after operation, even if large quantities of fluid are set free in the wound and absorbed. Sometimes, however, symptoms of this nature occur after operation, their appearance being usually delayed for thirty-six to forty-eight hours but lasting for some days. Urticaria is usually absent, and there is no absolute criterion whereby we can distinguish anaphylactic from ordinary post-operative shock; but all surgeons must have been struck by the frequency of unexplained post-operative reactions after hydatid operations, and there is no doubt that some of these are anaphylactic. In the past they have been explained, without any confirmatory evidence, as being due to reactionary pyrexia, operative shock, peritoneal reaction, post-anaesthetic vomiting, mild infection, etc. Grave symptoms, however, occasionally occur, and Dévé has collected a small series of fatal post-operative cases. It is probable that further careful observations on these or on other cases of hydatid disease will lead to the recognition of anaphylactic symptoms in more cases than is generally admitted.

SECONDARY ECHINOCOCCOSIS.

Rupture of a hydatid cyst or puncture by a trocar with evacuation of the specific fluid does not necessarily cause death of the parasite—probably it rarely does unless micro-organisms are introduced. The parasitic elements have such power of persistence that they can survive and ultimately develop into new cysts often at a distance from the original, this development being best described as secondary echinococcosis to distinguish it from

the development of cysts primarily from hexacanth embryos. For many years clinicians had noted the occurrence of multiple cysts, and although there were a few dissentients, these were regarded by the majority as due to multiple primary infestations. The history of the gradual recognition of the true nature of these cysts is of some interest. In 1793 the famous John Hunter, with characteristic acumen, first suggested that some abdominal and pelvic cysts were secondary to implantation of hydatid element from intraperitoneal rupture of a visceral cyst. Chareot and Davaine² criticized this view on theoretical biological grounds, and it was gradually forgotten until 1861, when Richard Bright,³ although he made no reference to Hunter's original suggestion, revived it and pointed out the dangers of puncture and operative interference. In 1877 Potain⁴ and Volkmann⁵ also supported the hypothesis, and warned surgeons against the risks of dissemination of the disease by these agencies.

From this time onwards many surgeons in all countries made clinical and operative observations which bore out this view, although some of them believed that multiple cysts were due to implantation of daughter cysts. In Australia, Graham, Watson, and MacCormack were the chief upholders of the doctrine of implantation of scolices. In spite of these direct surgical observations, the majority of pathologists and helminthologists, although they believed that—as was proved experimentally by Lebedeff and Andrejew⁶ in 1889—daughter cysts could implant, refused to countenance the view that such highly differentiated structures as scolices could, as it were, revert in their life-cycle. Even as late as 1900 such a view ranked with many authorities as a biological heresy, as it seemed contrary to all the laws of the development of the cestodes laid down by Van Beneden.

Further accumulated observations and experimental researches, however, have conclusively proved that such a retrogressive metamorphosis is not only possible, but that it is relatively common and of great clinical importance. Experimental work on the problem has been carried out and confirmed by d'Alexinsky⁷ and Dévé⁸ in Europe, Lagos Garcia in South America, and myself in Australia. Briefly, if washed living scolices are injected into rabbits by the subcutaneous, intramuscular, intraperitoneal, or intravascular routes, positive results with the production of secondary cysts are obtained in about 75 per cent of the experiments.

The vesiculation of the scolex is a relatively simple process. It becomes hydropic, its cells spread out on the inner aspect of the cuticle, which then rapidly thickens and becomes laminated. The only structures that persist are the hooklets which are found aggregated in a confused mass at one pole, but as the cyst grows they also become scattered and disappear. There is no doubt that many of the scolices are overwhelmed by the cellular reaction of the host, and undergo phagocytosis and fibrosis—a fact which is borne out by many clinical observations in the human subject. There seems little doubt that fragments of the original germinal membrane can also lay down protective laminated membrane around islets of nuclear material and also give rise to cysts.

From these and other correlated observations, therefore, has emerged the unique fact that such is the persistence of the parasitic elements that any

of them can, if shed into the tissues under aseptic conditions, implant and give rise to secondary cysts which may ultimately become fertile. Recognition of the frequency and importance of this has led to great advances in our conception of the pathology of hydatid disease.

Clinical Types.—There are many clinical examples of this phenomenon, as it can occur following practically any type of rupture, but reference will be made only to the localized, the peritoneal, and the metastatic types.

Localized Secondary Echinococcosis.—Rupture of cysts of the subcutaneous or muscular tissues, owing to their exposed position, is relatively common. As a result germinal elements, mainly in the form of young scolices, are shed into the tissues under aseptic conditions, and, although some of them no doubt are destroyed by phagocytosis, many survive and form multiple secondary cysts which by their growth often overwhelm the original primary cyst. They are comparatively uniform in size, and may be carried some distance by muscular action, particularly along perivascular and perineural sheaths. The name 'seed hydatids' which has been given to them is very appropriate. As is the case with most secondary cysts, degenerative changes with the production of cholesterol are common. Slight leakage of fluid from a simple cyst following puncture or mild trauma may give rise to a similar phenomenon within the confines of the adventitia—the fluid being absorbed and multiple cysts developing *in situ*.

This constitutes one of the commonest types of reactive daughter-cyst formation, and is the explanation of the reappearance of a multivesicular cyst, or cyst containing daughter cysts, at the site of a punctured or injured cyst. It is possible that the most logical way of looking at endogenous daughter-cyst formation may be to regard it as a special type of localized secondary echinococcosis—due to some complication which has not necessarily manifested itself clinically.

Post-operative recurrence in the original cyst, in the scar, or in the depths of the field of operation, constitutes another well-known type. Owing to the need of what are sometimes difficult and extensive operations to eradicate these recurrent cysts, this is often a serious matter, and until hydatid cysts are regarded as infective foci and respected as such, these cases will continue to be a feature of our records. They are nearly always due to the shedding of the easily missed scolices, and the method of preliminary sterilization by formalin introduced by D  v  , Qu  nu, and others, and now in general use throughout the world, should practically eliminate this sequela.

Secondary Cysts of the Peritoneum and Pelvis.—These are very common, and are always due to leakage of germinal elements from a primary cyst of the liver, or more rarely of the spleen, kidney, or omentum. A consideration of the possible sequelae of rupture of a hepatic cyst is of interest in that it illustrates the protean manifestation of hydatid disease in this situation.

Occasionally the adventitia alone gives way, the intact parasite being extruded, coming to rest, and gradually acquiring a new adventitia, somewhere in the abdominal or pelvic cavity. As no fluid is shed this event may occur quietly, with no anaphylactic or other symptoms and no resultant secondary cysts. This is an explanation of one type of primary pelvic hydatid cyst.

Usually, however, the cyst ruptures, hydatid fluid and scolices being shed into the peritoneal cavity with some degree of anaphylactic shock as described above. The scolices may be shed in thousands, and are carried by the rush of fluid, by gravity, or by intestinal movement, to various parts of the cavity—as a rule to the lower quadrants. They soon become surrounded by lymph and eosinophil leucocytes and so rapidly fixed to a new secondary site. Doubtless many of them are overwhelmed and undergo fibrosis; sometimes the peritoneal or omental reaction around these disintegrating scolices is so extensive that a pseudo-tuberculous appearance and thickening of the peritoneum are produced (*Fig. 169*). Many, however, survive, become surrounded by a new adventitia, undergo vesiculation, and develop into secondary cysts. Just as in the case of any other foreign body introduced into a serous cavity, fibrotic encapsulation follows, the peritoneal epithelium gradually spreading over all until the secondary cyst gives the appearance of having developed in an extraperitoneal situation. It was non-recognition of this simple process that led many of the older pathologists to believe that these cysts were multiple primary cysts developed from hexacanth embryos brought to the subperitoneal zone by the blood-stream, or by direct migration from the upper part of the alimentary canal.

These secondary cysts are of slow growth, so that there is always a latent period of from five to twelve years at least during which growth takes place until they in their turn produce symptoms. Owing to the rapid and complete recovery in most instances from the initial rupture, it is often misinterpreted by the clinician and its true significance is unrecognized until at a later date the discovery of multiple abdominal cysts leads to a retrospective diagnosis. It is thus not uncommon for this complication of intraperitoneal rupture of a visceral cyst to occur in youth, but for its clinical manifestation and recognition in the form of multiple abdominal cysts to be delayed till adult age is reached. Secondary abdominal cysts are always multiple, often irregular in shape and size, and owing to the relative tenuity of their adventitia are prone to further ruptures with repetition of the above effects. As a result, the whole peritoneal cavity may become filled with hydatid cysts in all stages of development—a very grave condition aptly described as hydatidosis.

It cannot be too strongly urged that, when multiple cysts are discovered in the abdomen or spleen, their secondary nature should be suspected and a search made for a primary visceral cyst. The original primary cyst may

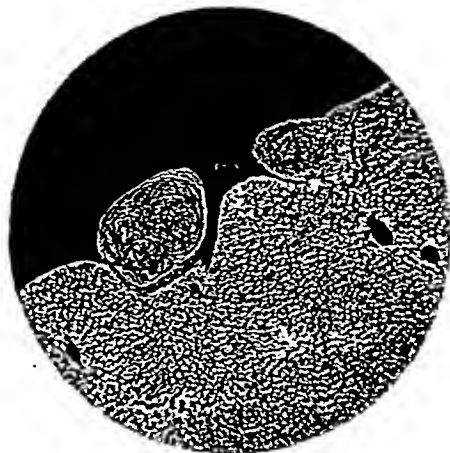


FIG. 169.—Formations of tubercles around disintegrating scolices on the edge of the liver following intraperitoneal injection.

be completely evacuated and replaced by fibrous tissue, so that after a lapse of years it may be impossible to detect the site except by very close examination. This is the explanation of some cases of obvious secondary abdominal cysts in which no primary cyst has been discovered at operation. Usually, however, the rent becomes occluded by adhesions and the residual germinal elements produce multiple daughter cysts—one type of localized secondary echinococcosis. It is a general rule, therefore, that when multiple simple peritoneal cysts are found there is also a primary visceral cyst containing daughter cysts. Somewhat similar phenomena to the above may occur following rupture of a cyst into the pleura or pericardium.

When a multivesicular hepatic cyst ruptures into the peritoneal cavity similar sequelæ occur, but owing to the frequency of communication with the biliary tract other possibilities arise. Although these are partly mechanical and partly infective, it seems appropriate to deal with them shortly here.

Because of the thickness of the adventitia and of the frequent presence of omental adhesions in these cases, rupture is less frequent and requires





Diagram	Type of rupture.	Immediate effects.	Effect on original cyst.	Late peritoneal effects.
	Polyspherical cyst with rupture of intact cyst.	—	Deep formation of abscess, biliary tract or suppuration very rare.	Simple abscess or sterile cyst. Chole-peritonitis or peritonitis is very rare.
	Unilocular cyst with rupture of mother cyst.	Aspiration, abscess and peritoneal abscess.	Complete communication, if abscess, sterile abscess of mother cyst, or smaller, biliary communication, infection or suppuration is very rare.	Multiple secondary peritoneal cysts very common. Pseudo-tubercles, one thickening to one chole-peritonitis or peritonitis is rare.
	Unilocular cyst with no biliary communication.	Aspiration, abscess, and peritoneal abscess.	Complete communication, if abscess, sterile abscess, or smaller, biliary communication is very rare.	Secondary peritonitis or sterile cysts due to daughter cyst, infection, sterile peritonitis, abscess, sometimes suppuration or abscess.
	Unilocular cyst with biliary communication.	Aspiration, abscess and peritoneal abscess, acute peritonitis.	Biliary fistula, secondary suppuration, abscess.	Suppuration frequent, secondary peritonitis, sterile abscess, choleperitonitis, or bile.

FIG. 170.—Schema of the possible sequelæ of intraperitoneal rupture of a hepatic cyst.

much greater trauma. As in the preceding type, hydatid fluid, débris, daughter cysts, and—if any of the latter are ruptured—scolices as well, are shed into the peritoneal cavity. There will be the same anaphylactic syndrome, the same risk of development of secondary cysts either from scolices or daughter cysts; but, in addition, bile in varying quantities may enter and lead to a choleperitonitis. This leakage of bile may occur at the time of rupture, or be delayed for some time until

a bile-duct ruptures owing to removal of cystic support. The flow may be very slight or profuse, and the results vary greatly. The peritoneum is at first extremely tolerant, a biliary ascites developing slowly over a period of months. As a rule, however, the collection of bile becomes localized by adhesions and more or less encysted. As is to be expected, the diagnostic difficulties are great and errors are very common. In spite of this tolerance of the peritoneum, infection is introduced sooner or later, and although this is usually of low virulence, the hand of the surgeon is ultimately forced owing to the development of local peritonitis. Occasionally infection occurs coincidently with the biliary leakage, in which case a subphrenic abscess or various degrees of peritonitis rapidly develop. These pathological sequelæ of intraperitoneal rupture of a hepatic cyst are summarized in Fig. 170.

Metastatic Secondary Echinococcosis.—This gives rise to the rarest but the most interesting type of all secondary cysts, and is due to a rupture of a

fertile simple cyst into the heart or venous system. This type of rupture may take place on the venous side of the circulation into peripheral veins or the right chamber of the heart, or into the arterial side, when it always takes place into one of the left chambers of the heart. As a result two sets of sequelæ are possible. In both, of course, grave anaphylactic symptoms may occur, although as a rule recovery takes place.

If the cyst ruptures into the venous side of the circulation, the scolices are carried through the right ventricle into the lungs, where they are filtered out and give rise to secondary pulmonary cysts (*Fig. 171*). These metastatic pulmonary cysts are characterized by their multiplicity, their bilateral and peripheral distribution, and by their uniformity in size.

In the second variety the rupture always takes place into the left auricle or ventricle, the hydatid elements entering the systemic circulation and giving rise to metastatic cysts in various parts of the body. Owing to the position and relative size of the carotid arteries, the majority of scolices are carried to the brain, which becomes the seat of the majority (60 to 70 per cent) of these secondary cysts. Some scolices, however, often escape to the kidney, spleen, or liver, and there give rise to secondary cysts. All these are simple cysts of approximately the same size, rarely becoming larger than a hen's egg.

The secondary cerebral cysts are in marked contrast to the single primary cysts of the brain in that they occur in the adult, while primary cysts occur classically in children. Indeed, primary cerebral cysts are very rare in adults—their frequency being only one-seventh of that in children. This is due to the fact that hydatid infestation usually occurs in children, and that cysts in this situation produce symptoms in a few years so that adult age is not reached.

The metastatic cysts are rare in childhood, because the cardiac cyst has to reach a certain size before rupture takes place—a matter of some years—and then the secondary cysts have to grow large enough to manifest themselves—also a matter of some years.

The original cardiac cyst may undergo obliteration by fibrosis, but as a

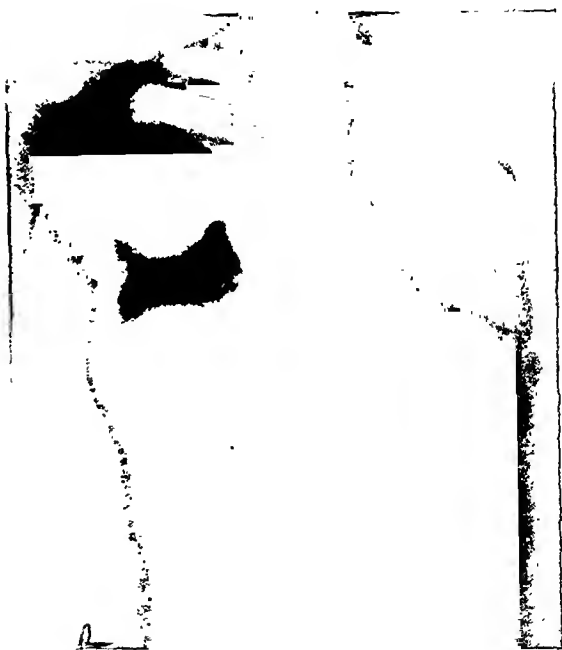


FIG. 171.—Radiograph of multiple metastatic pulmonary hydatid cysts.

rule some germinal elements remain therein, the rent closes, and reactive daughter-cyst formation occurs in the original site. Provided the patient does not succumb to the effect of the metastatic cysts—a not unusual event when the brain is involved—the primary cardiac cyst later ruptures a second time, with, as a rule, fatal embolism by daughter cysts.

The clinical picture of these cardiac cysts is of great interest, and owing to careful pathological studies the whole story can now be accurately told. It may be divided into six phases as follows:—

1. Period of some years (ten to fifteen) during which growth of the primary cardiac cyst with no symptoms takes place.

2. Rupture of the simple cyst into the pericardium, or more commonly into one of the cardiac chambers, with a dramatic anaphylactic syndrome which is often misinterpreted.

3. Phase of latency (two to five years) during which the secondary cysts develop in the pericardium, lung, brain, or other organ; the original cyst occasionally becomes obliterated by scar, but usually a multivesicular cyst containing daughter cysts re-forms in the primary site.

4. Rupture of one of the pericardial cysts may occur into one of the cardiac chambers, with repetition of phases (2) and (3).

5. Development of symptoms due to the multiple pulmonary or intracranial cysts, the latter often causing death.

6. If the patient lives long enough, a second intracardiac rupture of the now multivesicular primary cyst, with, as a rule, anaphylactic shock and death from daughter-cyst embolism of cerebral or pulmonary vessels.

It is therefore an important pathological point that the discovery of multiple pulmonary or cerebral cysts should direct attention to the heart, and vice versa.

These examples illustrate the classical manifestations of secondary echinococcosis, which is now clearly established as regards its etiology, pathology, and clinical aspects. It may be taken as a general rule, in the case of multiple cysts, that if the extrahepatic cysts are more than one-third of the total, it is probable that they are secondary cysts.

MECHANICAL EFFECTS OF RUPTURE.

The mechanical effects of rupture of a cyst are seen in cases of rupture into a natural excretory or secretory channel. Following the initial flooding with fluid, which is rarely serious, the passage of hydatid products along the channel leads to attacks of colic or intermittent partial or complete obstruction. These are as a rule more or less immediate effects, although they may continue for months as fractional evacuation of the cyst contents occurs.

It must be emphasized that micro-organisms do not find their way through an intact laminated membrane, and that in the case of simple cysts some degree of rupture of this membrane is an essential preliminary to suppuration. Once the hydatid membrane collapses, however, some serum is exuded into the cavity from the adventitia, and this with hydatid debris makes such an excellent pabulum for the growth of micro-organisms that when contact with a lining epithelium is made infection is usually only a

matter of time. There is no doubt, however, that small ducts or tracts may have at one time opened into the cavity and yet it may remain aseptic for years, and occasionally actual closure and repair of the opening may occur. Obviously the time that elapses between rupture and the introduction of infection must depend on a great number of variable factors, so that, although the introduction of infection may coincide with the rupture, it is usually delayed for weeks or months. As these two processes are closely bound up with each other they will be considered together in the following paragraphs.

Rupture of Hepatic Cyst into Biliary Passages.—Bile staining of multivesicular hepatic cysts is very common owing to the persistence of small bile-ducts in the adventitia and also the occurrence of previous leakage from the original simple cyst. As the cyst continues to enlarge, however, it may cause a quiet pressure necrosis of the wall of a large bile-duct, and if the opening becomes large enough some of the hydatid debris is forced into the duct. There is no doubt that such ruptures are relatively common, and I believe that some degree of intrabiliary rupture is the commonest of all the complications of hepatic cyst. This may be a mild quiet leak into the passage of fluid and small particles of debris, or a frank massive rupture with or without occlusion of the main ducts with daughter cysts. It is important to recognize that this rupture takes place most commonly into the intra-hepatic portion of the ducts, this being one of the reasons why its frequency has not been generally recognized. *Fig. 172* gives a schematic representation of the sites of this rupture.

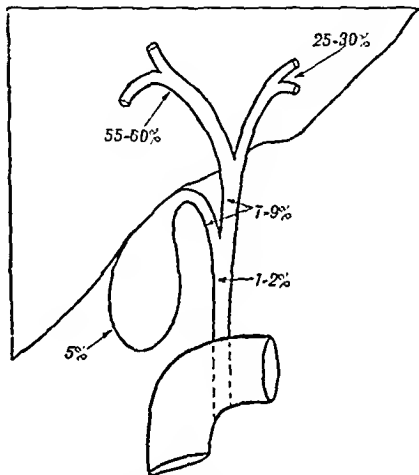


FIG. 172.—Schematic representation of the sites of rupture of hepatic cysts into the bile-ducts.

As a rule the biliary rupture takes place in cysts containing daughter cysts—according to Duprey in 93 per cent of a series of 55 cases collected by him. There seems little doubt that gross rupture, with the passage of collapsed or intact daughter cysts down the duct, is simply the culminating event of a series of minor ruptures. It is thus probable that the original rupture occurred years before, and owing to the maintenance of aseptic conditions and occlusion of the opening, reactive daughter-cyst formation occurred, giving rise to a multivesicular cyst in place of the mother cyst. Once rupture has occurred, the passage of the material down the ducts is helped by the intra-cystic pressure, diaphragmatic movement, and abdominal muscular action; and once started, the slippery non-irritating membrane is helped by the increased biliary flow after meals. In this way fractional evacuation of the cyst may take place into the duodenum. There may be, however, long periods of quiescence, so that months or years may elapse before all is evacuated.

The passage of débris or collapsed daughter cysts down the ducts, with varying degrees of pain, is the probable explanation of the pain experienced in most cases of complicated hepatic hydatid disease. This is a more reasonable explanation of the pain than pressure on the ducts, perihepatitis, hepatoptosis, or pressure on the cœliac plexus—explanations that have been advanced by clinicians in the past.

As a rule this is associated with biliary colic and transient or persistent jaundice; the biliary tree being at times markedly distended and the gall-bladder being found enlarged in 66 per cent of the cases. *Fig. 173*, for which I am indebted to Mr. B. T. Zwar, of Melbourne, illustrates the condition that



FIG. 173.—Radiograph obtained by the injection of 15 per cent sodium iodide into the biliary channels through a communicating hydatid cyst. (By courtesy of Mr. B. T. Zwar.)

is found in some of these cases. This radiograph was obtained by running 15 per cent sodium iodide solution into a left-sided hepatic cyst which extended into the right hepatic lobe, and which had ruptured into the biliary passages with the syndrome mentioned above. It shows very well the distension of all the biliary tree and the presence of the opaque iodide in the gall-bladder and duodenum.

In cases with this complication many puzzling variations of the pathological picture can occur—such as coexistent rupture into the bronchi, alimentary canal, or peritoneum, with consequent modifications of the clinical picture.

Owing to the presence of bile-stained débris in a cavity which is poorly drained, and to the frequency of attacks of obstructive jaundice, the patient is in imminent danger of severe or fatal complications. Suppuration is the most frequent concomitant of intrabiliary rupture, occurring in 60 per cent of all cases. There seems little doubt also that in the majority if not all cases of suppuration of hepatic hydatids some degree of intrabiliary rupture has occurred at one time or another. As is to be expected, this clinical condition simulates closely the migration of gall-stones, and the diagnosis of cholelithiasis is the commonest error in Australian surgical practice.

It is certain that, were it more generally recognized that hydatids not infrequently, and gall-stones comparatively rarely, involve the larger ducts, this diagnostic error would become less common.

The following points are of interest in the differential diagnosis:—

Age.—Hydatid disease involving the duets tends to occur at a slightly younger age than stone in the common duct. i.e., 30 to 40 rather than 45 to 55.

Sex.—In hydatid disease males are found affected just as commonly as females, whereas in gall-stone disease females are four times as commonly affected as males.

Pain.—The pain of hydatid disease is not so severe as true gall-stone colic, owing to the non-traumatizing nature of the membrane.

Previous History.—In gall-stone disease there is nearly always a comparatively long history of flatulence, etc., which is often completely absent in hydatid disease, although in the latter there may have been attacks of an anaphylactic nature.

Examination of the Stools.—Examination of the fæces is the most important clinical method at our disposal, and should be used as a routine in all cases of jaundice. It is easily carried out by washing the fæces in a stream of water, when the tough bile-stained laminated membrane, which resists putrefaction in a remarkable way, is often discovered.

Hepatomegaly.—This is much commoner in hydatid disease than in gall-stones.

Jaundice.—This is characteristic of hydatid disease involving the duets, being present in 80 to 90 per cent of the cases, while in gall-stone disease gross persistent jaundice is practically confined to cases of stone in the common duct.

Radiography.—This should be applied as a routine, as it may indicate subdiaphragmatic distortion or calcareous change in a cyst, while cholecystographic methods are of value in gall-stone disease.

Immunological Reactions.—These are of the utmost value, and the routine use of the hydatid complement-fixation test and the intradermal test of Casoni has revolutionized the diagnosis of hepatic hydatid in Australia.

With the onset of infection introduced by way of the biliary channels, the patient sooner or later, depending on the tension of the collection and the presence or absence of a valvular effect at the opening, shows evidence of intrahepatic suppuration and cholangitis, with rapid alteration of the clinical picture. It is a remarkable fact that in 15 to 20 per cent of the cases gas-producing organisms are present, and percussion and radiographic methods may detect the presence of a bubble of gas above the fluid in the cyst. In addition many other complications such as diffuse hepatitis, subphrenic abscess, peritonitis, empyema, and rupture into the bronchi may occur.

Hepato-bronchial Fistula.—This is not uncommon, and usually occurs in right-sided subdiaphragmatic cysts. The layers of the diaphragm and pleura are usually adherent from low-grade perieystic inflammation, and the cyst gradually penetrates through the lung and ruptures into the bronchus. When this happens the patient expectorates large quantities of daughter cysts and debris which is often stained with bile. It cannot be too strongly urged that the expectoration of daughter cysts, because of their rarity in purely primary pulmonary cysts, usually means that they originate in a hepatic cyst and that the sputum in these cases should be examined for bile. Sometimes the

cyst is non-infected at the time of rupture and fractional expectoration of the dependent cyst may proceed for months; but sooner or later the poor drainage and the presence of a large bronchial communication lead to the introduction of micro-organisms and suppuration.

All sorts of variations of this pathological picture can occur, such as rupture into the pleura with the production of a chylothorax, a pyochoylothorax, or a pyopneumochoylothorax. At other times localized gangrene of the lung occurs in the region of the communicating track, while at others there is a coexistent rupture into a large bile-duct or into the alimentary canal, causing puzzling symptoms. These cases constitute some of the most difficult problems in hydatid disease, and unless the surgeon is alive to the

possible vagaries of a large hepatic cyst, mistakes of diagnosis, treatment, and prognosis are inevitable.

In spite of the frequent proximity of cysts to the intestine, rupture into the alimentary canal rarely occurs. When it happens, evacuation with natural cure may follow; this depends on the size and site of the cyst and of the communication. There is, of course, a big risk of infective processes, and as a result the mortality in such cases reaches 50 to 60 per cent. In the same way rupture into the urinary tract may occur, with an almost exact simulation of renal colic due to stone. There is, as in the hepatic cases, the same attempt at fractional evacuation, the same intermittent blockage of the tract, the



FIG. 174.—Radiograph of simple pulmonary cyst before rupture.

same risk of infection. The latter, which is often of a low-grade type, is usually delayed for years, and seems to be more or less dependent on the degree of obstructive hydronephrosis that is induced.

Rupture of a Pulmonary Cyst into a Bronchus.—This is the natural end of most pulmonary cysts, because by the time a cyst reaches a diameter of four to five inches it nearly always comes into contact with a large bronchus, the erosion of which causes a small part of the cyst wall to become unsupported. Following a cough, a muscular effort, an injury—or even spontaneously—the cyst wall gives way and the cyst contents enter a bronchus. Although partial rupture, with the escape of small quantities of fluid, may be masked by the accompanying hæmoptysis and bronchial

secretion, as a rule a comparatively large amount of fluid rushes into the bronchus with dramatic results. Rarely the patient is asphyxiated by the fluid or by the impaction of membrane in the glottis. As a rule, however, the severity of the symptoms is out of proportion to the amount of fluid expectorated, and it is probable that the persistent frothy sputum, dyspnoea, cyanosis, and general collapse so universally noted, are in the main anaphylactic in nature.

After rupture the patient usually recovers rapidly, although for some time repeated expectorations of fluid and gradual evacuation of the cyst contents may occur.



FIG. 175.—Radiograph of empty pulmonary cyst thirty-six hours after rupture. Same case as Fig. 174.



FIG. 176.—Radiograph of ruptured hydatid of the lung ten days after rupture, showing formation of a pneumocyst. Note fluid level with gas above it. Same case as Fig. 175.

The patient may have attacks of coughing and hæmoptysis but remain afebrile and in good health throughout, while natural cure, with collapse of the cavity and fibrosis, proceeds. Natural cure in this way depends on many factors. The size of the cyst is important, as on it will depend the thickness of the adventitia and therefore the possibility of complete collapse, and also the time taken for disintegration and expectoration of the cyst wall. As is to be expected, small cysts and cysts in children more readily undergo natural cure. The larger the bronchial communication and the more dependent its position, the better are the chances of evacuation. Apical cysts are

more likely to involve bronchi while yet small, and the opening will probably be dependent then, facilitating drainage. In the same way deeply placed parabronchial cysts will fulfil most of these conditions, and all are agreed that these cysts should not be attacked surgically, and that if left alone often about 75 per cent will undergo natural cure.

In a great many cases the contents of the cavity offer an excellent pabulum for bacterial growth, and owing to the bronchial communication organisms find their way in. At first these are saprophytic, but sooner or later pathological organisms, at first of low virulence, but later virulent cocci and often anaerobic gas-producers, enter. The adventitia become thickened and lined on the inner aspect with a pyogenic membrane, and all the signs of intrapulmonary abscess supervene with modification of the clinical picture (*Figs. 174-176*).

Suppuration of a pulmonary hydatid can only be produced in this way, and owing to the risk of extension in the lung and to the pleura may lead to grave sequelae. Apart from these risks the prolonged toxæmia is of serious moment, and surgical intervention is usually required.

Hydatid Pneumothorax.—Another interesting type of rupture is that of a subpleural pulmonary cyst into the pleural cavity. This may be comparatively quiet, being associated only with immediate anaphylactic symptoms or the formation at a later date of secondary pleural cysts.

As a rule, however, the subpleural cyst is large enough to have involved a patent bronchus, in which case a pneumothorax follows the rupture. This hydatid pneumothorax, occurring as it does in a previously healthy patient, may be most dramatic in its onset. At first the effect is purely mechanical, although the mild irritation of the pleura often leads to a simple hydropneumothorax. In the majority of cases organisms are introduced from the patent bronchial opening and a pyopneumothorax develops. This may be delayed for days or weeks, but as a result the patient develops the signs of intrapleural suppuration.

It is obvious that this condition may be confused with tuberculous pneumothorax, but unlike that disease it is fairly common in adolescents; it is more common on the right side; there is rarely any suggestive previous history; there are no signs present on the opposite side; tubercle bacilli are absent from the sputum; anaphylactic symptoms are common; and the typical hydatid immunological reactions are often positive.

SUMMARY.

The various complications described above do not exhaust the possibilities, but they serve to illustrate the infinite variety possible, and to emphasize the fact that rupture, the formation of secondary cysts, and suppurative effects all combine to make hydatid disease a dangerous one for the patient and one full of problems for the clinician. In conclusion, the following facts, which are not always given sufficient prominence in discussion on the pathology of this disease, are reiterated :—

1. The majority of hydatid cysts are nearly as old as the patient harbouring them: complications increase in frequency as age advances, and practically all depend on some degree of rupture.

2. Hydatid anaphylactic symptoms are much more common than is usually recognized, and are evidence of leakage from the cyst.

3. Secondary echinococcosis is relatively common, and all cysts containing scolices must be regarded as infective foci and potential sources of dissemination of the disease.

4. Intrabiliary rupture of hydatid cysts of the liver is one of the commonest complications of hydatid disease in this situation, and the simulation of gall-stone disease may be very accurate.

5. Suppuration in hydatid disease is a severe complication and is always preceded by some degree of rupture.

I have to acknowledge the courtesy of the Australian Medical Publishing Company, of Sydney, for permission to reproduce several of the illustrations.⁹

REFERENCES.

- ¹ DÉVÉ. F., *Rev. de Chir.*, 1911, July, 513.
- ² CHARCOT and DAVAINE. *Mém. Soc. de Biol.*, 1852, 103.
- ³ BRIGHT, R., *Clinical Memoirs on Abdominal Tumour*, 1861, 56. London.
- ⁴ POTAIN. *Gaz. des Hôp.*, 1877, 761.
- ⁵ VOLKMANN, 6th Congress of Surgery, Berlin, 1877.
- ⁶ LEBEDEFF and ANDREJEW, *Arch. f. pathol. Anat.*, 1889, cxviii, 552.
- ⁷ D'ALEXINSKY. *Arch. f. klin. Chir.*, 1898, 796.
- ⁸ DÉVÉ. F., "Echinococcose secondaire", Thesis, Paris, 1901.
- ⁹ DEW, H., *Hydatid Disease*, 1928. Sydney: Australian Medical Publishing Co.

THE TREATMENT OF INFECTED OPEN FRACTURES.*

By ERNEST W. HEY GROVES,

PROFESSOR OF SURGERY AT THE UNIVERSITY OF BRISTOL.

THE importance of this subject can scarcely be exaggerated. Not only is this type of injury becoming more common every day, owing to the increasing prevalence of machinery and road accidents, but also the results of treatment are often extremely bad in respect of loss of life, limb, or function. Further, a consideration of many of these bad results inevitably leads to the conclusion that treatment has been at fault and that lives, limbs, and function have been needlessly sacrificed.

There is a very real danger of the problems concerned being dealt with inefficiently, simply because they have become common or uninteresting. It is necessary to remind ourselves sometimes, when absorbed in the romance of modern surgical and scientific advance, that we must not neglect the common every-day injury, for by doing so we may cause the loss of life or limb just as certainly as if we failed to diagnose or treat some obscure disease of the brain or abdomen. Our responsibility in this matter is a very grave one, because the correct and timely treatment of an infected fracture can definitely save a life or limb, whereas the most intensive study of visceral disease will often only serve to mark the progress of an incurable malady.

Mistakes in the Treatment of Infected Fractures.—There are three common types of mistakes into which we are likely to fall in this matter. They are those of neglect, delay, or error of judgement. An injured man is brought to the casualty department of a hospital and is attended by a very recently qualified house surgeon. The wound is washed and painted with iodine, and the limb put on to a splint. The patient is then admitted to the ward, and very often nothing more is done until the next day, when a skiagram is taken. Such a routine really amounts to neglect, and would be considered so if the patient suffered from a punctured wound of the head, chest, or abdomen.

The fracture case may not be seen by an experienced surgeon for a day or two unless some grave symptoms arise. After a delay of one or two days the case is first seriously considered and a definite line of treatment adopted. Then if the skiagram shows badly placed bones or much comminution, the surgeon may be tempted to adopt some drastic treatment—for example, amputation, the plating of the fracture, or putting up the whole limb in plaster-of-Paris so as to hide the horrid sight under a white covering.

It must not be thought that in referring to these types of mistakes I am making an *ex-cathedra* denunciation of other people's sins. I am speaking of

* Introduction to a discussion on this subject by the Association of Surgeons of Great Britain and Ireland in Bristol, on May 1, 1930.

things which have been done by myself or in my service, for which I am sorry, and which I hope will not be done again.

Experience shows that in the avoidance of these common mistakes three broad principles should be observed :—

1. Every infected fracture should be treated with the same care as would be given to a case of penetrating wound of the head or abdomen. The full surgical team should set about the problem, the pathological and radiographic departments giving the necessary co-operation.

2. This extensive examination and treatment should take place at once, with no more delay than is permitted in the case of a ruptured gastric ulcer.

3. The ultimate treatment of the fracture should be carried out in such a way as to assist and not endanger the primary object of curing the infected wound. Unless or until infection has been cured, drainage must be provided, and the infected tissues must not be loaded with foreign bodies. The limb must be put up in such a way as to give every assistance to free circulation of blood and lymph.

Results of Treatment.—The consideration of statistics of the results of open fractures is made difficult by many factors besides the obvious ones which beset all statistical investigations. Two of these difficulties may be mentioned. It is impossible to group together various bones for the purpose of the inquiry. We cannot compare events happening to a metatarsal bone or rib with those occurring in the femur. Further, it is quite impossible to have any measure of the degree of the original infection of the fracture, and it would be obviously absurd to compare results of a fractured tibia when a sharp fragment of bone just perforates the skin with those when the bone is crushed and the soft parts are cut by a dirty cart-wheel. In order to eliminate these two factors I have collected the last cases which have come under my own personal investigation of fractures of the tibia and fibula in which the wound was caused by direct external violence.

I have careful records of 50 consecutive cases of infected open fractures of the tibia and fibula, which show the following results :—

		Deaths (following amputation) ..	2	Per cent	4
Amputation	8	16	
Good results	34	68	
Poor results	7	14	
Non-union	1	2	
			50	100	

These cases may be divided into recent cases, where we were responsible for the treatment from the outset, and old cases sent to us one or more weeks after the accident.

			Recent Cases	Old Cases
Deaths (following amputation)	2	—
Amputation	5	3
Good results	26 (74.3%)	8 (53.3%)
Poor results	4	4*
			35	15

* Including 1 non-union.

Death was due to gas gangrene (1 case) and streptococcal septicæmia (1 case). Apart from these cases, amputation was called for on account of damage to blood-vessels (secondary hæmorrhage, 1 case) or gangrene. Good results were obtained in three-quarters of the recent cases, and in about one half of the old cases.

Of the causes which contribute to these results four stand out for consideration: (1) The concurrent injury to the circulation producing traumatic gangrene; (2) An overwhelming virulent primary infection—these two causes of failure are inevitable and outside our power to control; (3) Delay in efficient primary treatment; (4) Faulty treatment of the fracture. It is these last two causes of failure which merit our close attention, because they are both under our direct control.

Delay in Efficient Primary Treatment.—This is perhaps the point of greatest practical importance, because it accounts for three-quarters of the failures and because it is due so often to sheer carelessness or procrastination. The common history of the case is this. A patient has a motor accident in some remote country place. He is taken to a nearby cottage hospital or doctor's surgery and the so-called first-aid treatment is applied. He is either detained for a few days or sent on by an ambulance to a city hospital, but in any case there is no serious treatment of the infected wound until one or two days have passed. It is very remarkable that at many country or cottage hospitals in which a perforated ulcer or acute appendicitis would be immediately recognized and operated upon, the infected limb wound is allowed to go out with nothing more than the most perfunctory treatment, although the opening up of such a wound with excision of the devitalized tissues is a matter of the utmost urgency and one which presents no great danger or difficulty.

Faulty Treatment of the Fracture.—The causes of failure under this heading are the faults of doing too little or the faults of doing too much. Doing too little in relation to the fracture is represented by the policy of simple splinting of the limb whilst the wound treatment is being carried out without any serious attempt to secure reduction of the fragments. This policy will certainly lead to gross malunion, which it will be very difficult to correct when the soft parts have healed.

The fault of doing too much is represented by the policy of indiscriminately removing bone fragments, and the even more dangerous practice of plating the septic fracture. Either plan involves grave danger of disaster: in one case by leading to non-union, and in the other to extensive necrosis.

The Treatment of the Infected Wound.—I feel sure that the guiding principle in these cases should be to regard them primarily as seriously infected wounds complicated by a fracture, and not as fractures complicated by a wound. There are two factors essential for effective treatment—timeliness and thoroughness. The sooner the better, and certainly within twelve hours of the accident, the patient must be anesthetized in the theatre with all the skilled assistance used for any serious emergency. Excision of the edges of the skin wound, enlargement sufficient to allow full and deep exploration, removal of foreign bodies, any fragment of bone which certainly has no soft tissue connection, and careful cutting away of damaged tissues—

these are details familiar to every student of war wounds, and there is neither reason nor justification for varying them in the treatment of infected fractures in civil life.

Three points require further reference: X rays should be used before the primary wound treatment, if this will not involve delay. Their use may be of great value in revealing foreign bodies or in indicating the types of fracture. The use of antiseptics is certainly advisable. Either flavine (1-1000) or tincture of iodine followed by alcohol will be efficient, but their use is merely accessory to the removal of grossly infected tissues and can in no way be a substitute for it. The choice between primary, delayed primary, and secondary suture must be made according to circumstances. In favourable cases primary suture should be carried out, but even in these a deeply placed wick of vaseline gauze will be a measure of safety against residual infection. Material from the wound will be sent for bacteriological report, and prophylactic injections of tetanus and anti-gas serum will be given.

The Treatment of Comminuted Fracture Fragments and of Bare Bone.—The utmost conservatism should be observed in the treatment of loose pieces of bone and of the ends of the main fragments, which may be bare. Bits of bone which have no attachment to soft parts should be removed; but this is a very different thing from pulling on every piece of bone that can be seen or felt in order to find out if it will come away. Exposed bare bone should generally be left alone, though it is sometimes wise to remove awkward points which may press upon the skin or soft parts.

The Late Treatment of Infected Wounds.—If primary treatment has failed to get rid of the infection, or if the case comes for treatment two or three days after the injury, then—assuming that mechanical cleansing has been carried out—the question of drainage, irrigation, or packing will have to be decided. The Carrel-Dakin method should be reserved for bad cases with deep complicated wounds, as it is difficult to carry out efficiently for an occasional case. In the majority of cases daily irrigation with hydrogen peroxide, with the provision of drainage by gauze wicks soaked in paraffin or by rubber tissue, will meet the necessity of the mechanical cleansing and removal of discharge. At the end of a month or six weeks, if the discharge is still profuse, a search must be made for sequestra. In this the X rays will be the greatest value, because they will reveal the dead bone as dense shadows in contrast to the rarefied living bone.

The Primary Treatment of the Fracture.—Although I am strongly convinced that the treatment of the fracture should be secondary to that of the infected wound, yet from the very outset the fracture must be correctly treated, first in order to favour wound healing, and secondly to facilitate the final mending of the bone. All that is necessary to do for the fracture in the first stages of treatment is to secure immobilization and correct alignment. If fixation of the fracture is not attained, obviously the irritation caused by movements of sharp splinters of bone against the soft parts will cause effusion and inflammation, and make the very conditions which maintain and increase the infection.

The limb should be slung up in a Thomas or cradle splint with sufficient traction to maintain correct alignment. At this stage it is better to use

adhesive appliances for traction, and to postpone skeletal traction for the time being. It is neither necessary nor desirable to exert any great traction force at this stage, because the soft parts will heal more kindly if they are not subjected to great tension. The first stage in the fracture treatment is limited to about one week, during which time the wound will be well on its way to healing either by first or second intention.

The Final Treatment of the Fracture.—Within a few days of the injury, and directly the infection has been overcome, the fracture must be put up in such a way as to secure full-length and good alignment whilst allowing continuance of the wound treatment. For this the one safe and efficient method is skeletal traction. The general procedure and the advantages of this method are so well known that it would be waste of time to describe it in any detail, and reference will be made only to certain points that are worthy of emphasis.

I have never been converted to the idea of the ice-tongs caliper; it is liable to become displaced during manipulation, it tends to drag, and is much more painful than a transfixion pin. For fractures in the lower end of the femur with wounds near the condyles it is much better to use tibial transfixion; only a $\frac{1}{8}$ -in. pin is required, and it can be left in place for six to eight weeks without harm. For fractures of the tibia and fibula, especially in the lower third, skeletal traction from the heel is of the utmost value. I know of no kind of footpiece which can hold and give traction for any length of time without damage to the skin. Whether the calcaneum is transfixed or whether the pin is passed above the heel bone is not very important. But if the supra-calcaneal transfixion is used, it is essential that the pin should be firmly fixed to the foot so as to prevent lateral rocking, which by dragging on the soft parts causes great pain and ulceration. The simplest way of securing this fixation of the pin is by a plaster bandage; otherwise it is necessary to use a special footpiece in which a sole-plate is clamped to the ends of the transfixion pin.

One more point is worthy of reference, and that is the great value of skeletal traction for complicated open fractures in the upper limb. In septic fractures of the humerus, particularly at its lower end, the arm is slung by the hand with the elbow at a right angle, whilst the traction is made by a pin through the olecranon. In fractures of the forearm bones steel nails of suitable length should be driven into the lower ends of the radius and ulna, and the arm suspended by a cord loop tied to the projecting heads of these. This will not only produce traction but will also exert a lateral pull on the bones, which counteracts the natural tendency they have to falling in towards one another. Difficult cases of fracture of the metacarpals and phalanges should be treated by traction through a small pin passed right through the base of the terminal phalanx, a stout needle serving this purpose very well.

Space will not permit a detailed discussion of other forms of treatment, but brief reference must be made to two particular methods.

The Plating of Infected Fractures.—It would seem almost unnecessary to mention this to an enlightened audience; but unhappily it is a practice which is only too commonly pursued. The great pioneer of bone-plating always insisted on the necessity of a clean field. In the War the disastrous

results of plating open fractures were proved by repeated tragedies. But still this practice continues, and I cannot allow this opportunity to pass without saying quite deliberately that I regard the plating of an open fracture as a surgical crime. It would seem that the jagged ends of a bone seen in an open wound exercise a sort of fatal fascination upon a certain type of adventurous practitioner who cannot resist the lure of the carpenter's tools. The septic bones are exposed still further, drilled, and screwed—and then the patient will be fortunate if after an extensive necrosis he escapes with his limb and his life.

The Use of Plaster-of-Paris.—In a general way, and until quite recently, I have been accustomed to regard the routine use of plaster-of-Paris in the treatment of open fractures as unjustifiable either in theory or practice. But first by reading Dr. Winnett Orr's writings, and then by meeting him and discussing the matter with him and those who have used his methods, I have been so much impressed by the value of his work that I determined to bring his ideas before this association, and now fortunately we have been able to do even better and bring Dr. Winnett Orr himself to expound his own views. Dr. Winnett Orr's method consists in immediate cleansing of the wound, packing it with sterile vaseline gauze, leaving it wide open, and, having adjusted the fracture, putting up the limb in a plaster case which is not taken off or opened for several weeks unless there is a rise of temperature or other untoward symptoms appear.

I am quite convinced that this method is of great value in special cases, but am not at all clear yet whether it would be wise to suggest it as a routine treatment. Its outstanding merits are that it saves the patient a great deal of suffering and a prolonged stay in hospital. It appears to have three obvious dangers and drawbacks. It is a dangerous method to put into the hands of an inexperienced practitioner. If the wound has been sterilized at the first operation, the leaving a wide open wound to heal by granulation is much less ideal than closing it by delayed primary suture. And, thirdly, it must in many cases tend to make the correct alinement of the fracture faulty, because this has to be controlled by a plaster case, which must become loose as the swelling of the limb goes down. However, perhaps after we have heard Dr. Winnett Orr, these objections may not seem to be so weighty.

NOTE OF A CASE OF INFECTED OPEN FRACTURE TREATED BY DR. WINNETT ORR.

On May 3, before a number of Fellows of the Association of Surgeons at the Bristol General Hospital, Dr. Winnett Orr carried out the treatment of a case which had been under my care. I have been so much impressed with his method and its results in this case than I cannot do better than describe it.

Alfred S., age 55, suffering from an open fracture of the right tibia and fibula caused by a fall from a roof on Feb. 27, 1930.

He was treated by immediate toilet of the wound, which was sutured, and he was put up on a back splint and side-pieces. However, the suturing broke down and a chronic suppuration ensued. Later on the leg was slung on a cradle splint

with pin traction from the calcaneum. After two months his condition had not undergone much change, and the X ray showed some necrosis of both fragments of the tibia, with considerable overlapping (*Fig. 177*). The wound was being dressed daily. The patient was in this condition when Dr. Orr saw him and operated upon him. He already had a pin through the os calcis. By means of this the foot was held firmly in the footpiece of a Hawley's table and sufficient traction applied to pull the leg out to a little more than its normal length.

OPERATION.—The wound, which before treatment had the appearance shown in *Figs. 178, 179*, was now excised and eurented. The overlapping bone fragments were chiselled away so as to leave a funnel-shaped cavity leading down to the deep surface of the



FIG. 177.—Skiagraph of leg two months after accident, showing bad position and necrosis.



FIG. 178.—Condition of wound on May 3, 1930, just previous to treatment by Dr. Orr.

tibia. A few small necrosed fragments were removed. Although the bone had been drawn out to rather more than the full length, considerable lateral displacement still persisted. This was corrected and maintained by means of a long

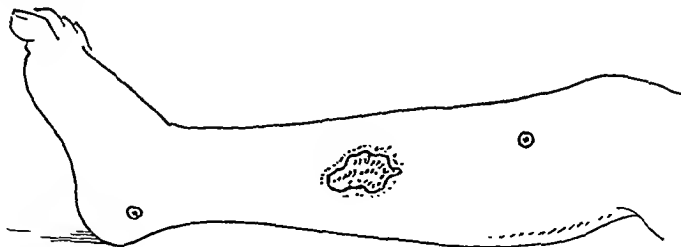


FIG. 179.—Diagram of relative size and position of wound and the sites of transfexion.

transfexion pin which emerged obliquely from the front of the wound. The wound, which had bled very freely, was packed first with dry gauze, then with iodine, and after with spirit. The bleeding was thus controlled, and into the dry cavity gauze impregnated with vaseline was packed firmly until it reached the surface.

TREATMENT OF INFECTED OPEN FRACTURES 301

Over the surface another vaseline pad was placed, and then a thin layer of cotton-wool. A long transfixion pin was passed through the crest of the tibia just below the tubercle. A plaster-of-Paris case was put on the leg so as to include the three transfixion pins, and extending to the lower third of the thigh. The patient was returned to bed upon an open-air balcony.

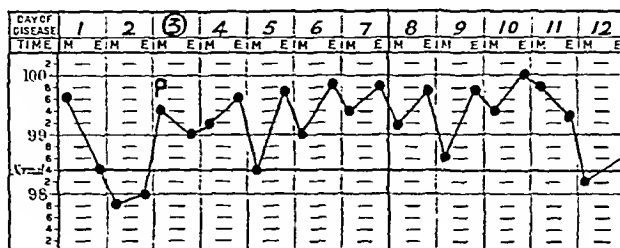


FIG. 180.—Temperature chart. P, Winnett Orr operation; plaster applied.

SUBSEQUENT PROGRESS.—This very drastic treatment has been followed by a perfectly smooth recovery. The man had a certain amount of pain and rise of temperature for the few days following the operation (Fig. 180). The pin which fixed the site of the fracture was removed after three weeks, and the other two pins a fortnight later. After the first week the plaster, which was deeply stained with blood, became rather odorous, and this continued all the time until its removal. The smell was not very objectionable in this case because the man was all the time on an open-air balcony, but it would have been much more unpleasant if he had been in a closed ward. (Figs. 181, 182.)

Eight weeks after it had been applied the plaster was cut off. The condition of the limb was very remarkable. All over the front of the leg was a mound of vaseline gauze mixed with stale blood and pus, and smelling very disagreeably. But on cleaning away this mess a small healthy granulating wound was found on which were a few minute bone fragments (Fig. 183). The limb was re-sterilized, the wound covered with a vaseline pad and again put up in plaster, below the knee but including the foot, for five weeks, the patient being allowed to walk with crutches. The X ray showed that union had taken place (Fig. 184). The man is now (Aug. 20, 1930) walking with side-irons from the knee to the shoe, and has good movement in both knee and ankle.



FIG. 181.—Skiagram after treatment, showing pins in the tibia.

To me this case has afforded a most convincing demonstration of the value of Orr's method. It has made good the claim of its author of greatly



FIG. 182.—Photograph of the limb in plaster, which was not changed for eight weeks.

simplifying the after-treatment. During thirteen weeks the patient was dressed only twice instead of the ninety-one times which the general routine of a daily dressing would have involved. But much more than this, the limb was restored to its full length with good alinement even in a case in which primary treatment had failed and chronic sepsis had been firmly established.



D. RILEY.

FIG. 183.—Appearance of the wound eight weeks after treatment on removal of the first dressing.



FIG. 184.—Skiagram of leg eight weeks after treatment. Note good union and position.

AN OPERATION FOR THE COMPLETE EXPOSURE OF THE ELBOW-JOINT.

By H. W. L. MOLESWORTH,

ASSISTANT SURGEON AT THE ROYAL VICTORIA HOSPITAL, FOLKESTONE.

EXPOSURES of the elbow-joint for the excision of tuberculous joints and severe open fractures by the classical methods associated with the names of Langenbeck, Kocher, and Cheyne are adequate for the remedy of the destructive lesions for which these great surgeons designed them. For more delicate



FIG. 185.—Anatomy of the elbow-joint. A, Ulnar nerve; B, Triceps; C, Olecranon, trochlear surface; D, Capsule; E, Flexor carpi ulnaris (olecranon head); F, Pronator teres (deep head); G, Epicondyle; H, Internal lateral ligament (cut); I, Pronator radii teres; J, Brachialis anticus; K, Nerve to pronator teres (median); L, Superficial veins; M, Anastomotica magna; N, Median nerve; O, Palmaris longus; P, Brachial artery. In this dissection the internal lateral ligament was cut through. This is not necessary in order to expose the joint.

operations, such as the removal of loose bodies, arthroplasty, and the operative repair of intra-articular fractures, the classical routes are somewhat too destructive, in that muscles are extensively stripped from their bony attachments, and complete exposure is only attained after the removal of parts of

the articulation. Limited arthrotomies (1) above the head of the radius, (2) by division of the olecranon process and its subsequent fixation with a screw, make it difficult to inspect the whole joint, in that the nature of the joint hinders a ready transference of vision and manipulation from the front to the back of the joint, and vice versa.

The operation to be described was suggested by experience with a patient who sustained a backward dislocation of the elbow, fracture of the internal epicondyle, and complete division of the ulnar nerve. The epicondyle was displaced within the joint, whither it had carried the proximal end of the ulnar nerve, and there was outward dislocation as a consequence.

Exposure of the ulnar nerve by the method suggested by Mr. Harry Platt discovered an open joint, and it was apparent that but a few touches with the knife were needed to dislocate the joint, or rather to open it on its inner side.

Anatomy (*Fig. 185*).—The key to the anatomy of the inner side of the elbow is the epicondyle with the group of muscles attached to it, and with

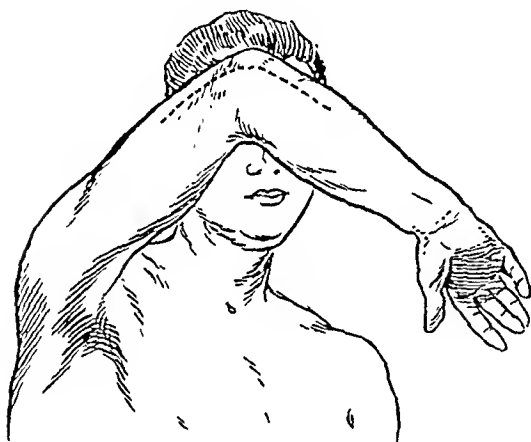


FIG. 186.—Position of arm and line of incision.

the strong band of the internal lateral ligament which runs from the epicondyle to the coronoid process of the ulna. These muscles—flexor carpi ulnaris (humeral head), flexor sublimis digitorum, palmaris longus, flexor carpi radialis, and pronator radii teres (humeral head)—strengthen the joint on its inner side. Neither the muscles nor their nerve-supply are compromised by detaching the internal epicondyle. The joint surfaces are not damaged by this manoeuvre, and the detachment is one which lends itself to repair. The ulnar

nerve is in danger on this side of the joint, but by making it a landmark, by clearing it and its branches through the extent of the field of operation, and by transplanting it to the forearm, it is safer than in the Langenbeck operation.

The median nerve is not exposed normally; if it is required for inspection, it may readily be found lying on the anterior surface of the brachialis anticus, or between the heads of origin of the pronator radii teres. Important blood-vessels are not encountered; the anastomotica magna branch of the brachial may be seen in the upper part of the incision in close contact with the brachialis anticus muscle. The posterior ulnar recurrent lies in the groove behind the internal condyle, where it is readily controlled. The anterior ulnar recurrent lies in close relation to the brachialis muscle, and is turned aside with that structure.

The Operation (*Figs. 186–190*).—The forearm is acutely flexed and fully

pronated; the elbow is raised, and rotated so that the inner side of the joint presents towards the surgeon standing on the side of the arm to be explored, and the outer side towards the patient's head. The incision follows the line of the ulnar nerve about four inches in the forearm and four inches in the upper arm (Platt) (*Fig. 186*). The ulnar nerve is sought in the arm just behind the internal intermuscular septum, and between that structure and the inner head of the triceps. It is gently freed as far as the groove behind the inner epicondyle. The space between the humeral and olecranon heads of the flexor carpi ulnaris is next sought, and these heads are separated; this discloses the nerve in the forearm. The branches to the muscle are mobilized and preserved; the branch to the joint given off behind the epicondyle is sacrificed. The fascia over the nerve in the groove is divided and the nerve is displaced into the forearm.

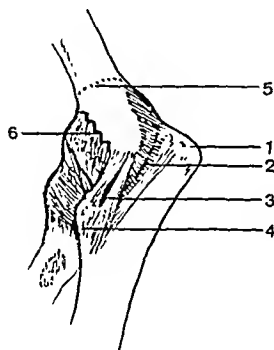


FIG. 187.—1, Insertion of triceps; 2, Incision in capsule; 3, Strong ligament, which is preserved; 4, Insertion of brachialis anticus; 5, Line of emergence of osteotome; 6, Line of detachment of capsule.

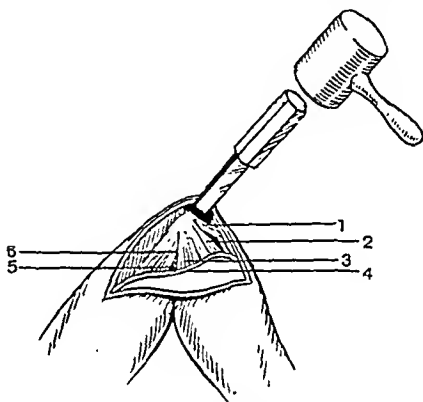


FIG. 188.—1, Anterior fibres of the internal lateral ligament; 2, Flexor carpi ulnaris (humeral head); 3, Pronator radii teres (humeral head); 4, Ulnar nerve; 5, Median nerve; 6, Internal intermuscular septum.

To this point the operation is that attributed to Stiles, and described by Mr. Harry Platt. The outline of the trochlea seen through the capsule is the next landmark, and the joint is opened by a short incision to the inner side of this process (*Fig. 187*). The incision is placed so as to be parallel to and behind the ligamentous fibres which run from the epicondyle to the inner margin of the coronoid process. Through this incision an osteotome is introduced (*Fig. 188*), pressed against the under surface of the epicondyle where it abuts on to the inner non-articular face of the trochlea, and the epicondyle is divided in an upward direction. (It is of advantage to drill a hole through the epicondyle from its tip upwards, outwards, and slightly forwards, to take the screw, before detaching this process.)

Next, find the outer border of the pronator radii teres, and clean this as far as the epicondyle, where a few fibres arise from the internal intermuscular septum, taking care not to injure the long twig from the median nerve which enters the deep surface of this muscle. Detach the internal intermuscular septum from the epicondyle, and turn the common flexor origin downwards towards the forearm. (Should it be desired to see the median nerve, it may

be sought in the interval between the humeral and ulnar heads of the pronator radii where it enters the forearm; the twig to the pronator leaves the trunk of the nerve at a higher level, and must be preserved.

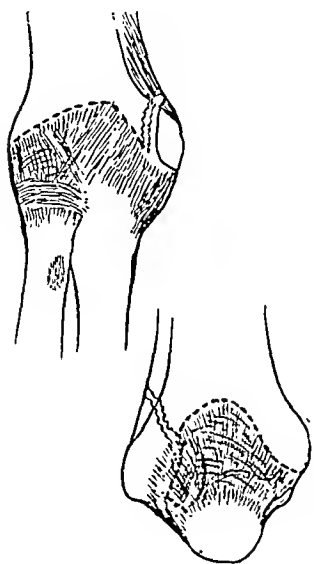


FIG. 189.—Diagrams showing extent of detachment for complete exposure. For exposures on the inner side less is necessary. The attachment of the external lateral ligament is preserved in all cases.

Returning to the joint, a sharp rugine readily detaches the posterior fibres of the capsular ligament from the margin of the olecranon fossa (Fig. 189). If in this manipulation the operator keeps close to the bone, the posterior communicating branch of the anastomotica magna artery is not encountered; it crosses about three-quarters of an inch above the attachment. The communication with the posterior ulnar recurrent artery will be severed, and needs a ligature at each end.

Retract the muscles to the flexor side of the internal intermuscular septum; the brachialis anticus and biceps with the median nerve and the brachial artery are all included in this retractor, the last three being seen only if the brachialis is cleaned rather more than is necessary. With a rugine detach the anterior capsular fibres of the joint from the coronoid fossa after the elbow has been somewhat extended. The joint is now free to the inner side, in front, and

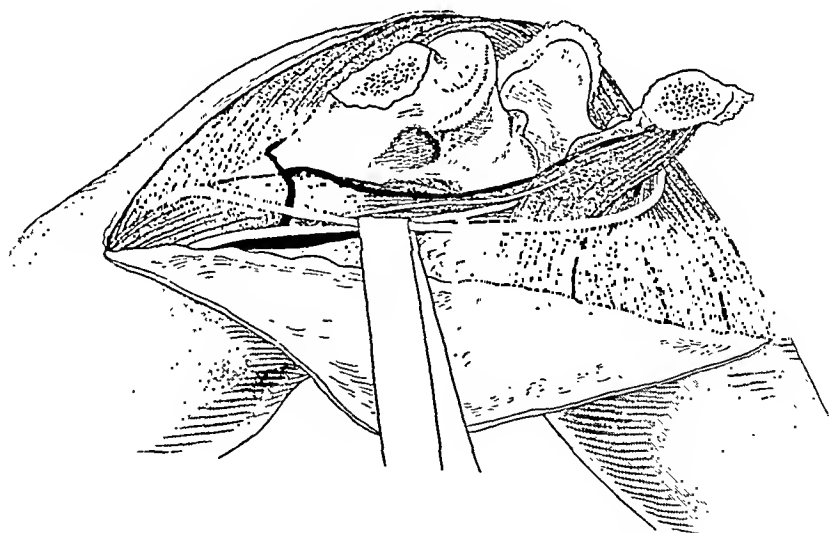


FIG. 190.—Diagram to show the extent of the exposure obtained.

behind; all that is needed is to flex the forearm to a right angle and strongly over-pronate it. The whole of the lower end of the humerus is

COMPLETE EXPOSURE OF ELBOW-JOINT 307

exposed to view, and the whole of the sigmoid fossæ on the olecranon, and with a little retraction the head of the radius and the semilunar synovial folds between this and the capitellum are also exposed (*Fig. 190*).

When the intra-articular manipulations are completed, the forearm is supinated to re-engage the joint surfaces. The epicondyle is fixed in its original position with a screw about an inch and a quarter long. A few points of suture unite the triceps to the brachialis, including in their bite the inter-muscular septum; the short incision in the capsule is closed, and the ulnar nerve is secured under the deep fascia of the forearm in an incision made to receive it. As an alternative method of disposing of the ulnar nerve, it may be placed with the median nerve between the two heads of the pronator radii teres, and in front of the epicondyle.

It is suggested that this operation, which is easily executed in practice, will prove of service in a search for loose bodies and fragments of bone, that repair of T-shaped and other articular fractures will be facilitated, that arthroplasty with preservation of the strong lateral ligaments may be expected to yield a stable joint, and that it lends itself to the exposure of those complicated injuries involving bone, joint, and nerves met with in this situation.

HÆMANGIOMA OF THE KIDNEY.

By W. ARTHUR MACKEY,

ASSISTANT TO THE PROFESSOR OF PATHOLOGY, GLASGOW UNIVERSITY.

HÆMANGIOMA of the kidney is of relatively rare occurrence, and authors of wide experience frequently report only a single new case. Infrequent though it may be, it is a condition of surgical importance, for it appears that a large proportion of cases ultimately demand operation more or less urgently. In the cases published an accurate pre-operative diagnosis has seldom been made. It seems desirable, therefore, to record a further case, in which sudden and severe hæmaturia necessitated immediate operation, the origin of the bleeding being a cavernous hæmangioma. In view of the difficulty experienced in distinguishing in the literature true angiomatous tumours from other lesions causing hæmaturia, an attempt has been made to review and classify the cases previously reported.

CASE REPORT.

HISTORY.—Miss A. M., age 61, had an attack of pain in the right lumbar region on March 13, 1929. This was of a dull, aching character and increased steadily in severity. On the following day she had a profuse hæmaturia accompanied by typical renal colic and the passage of worm-like clots. Attacks continued for the next four days, at the end of which X-ray plates of the right kidney region were taken, without revealing stone. She was seen by Mr. Roy F. Young on March 18, when severe hæmaturia was still present. The general health was good, and symptoms of renal disease had never previously been observed.

ON EXAMINATION.—The patient was healthy in appearance. Some tenderness in the right lumbar region was the only sign of renal involvement; no enlargement of the kidney could be detected. The urine was acid and heavily mingled with blood.

Cystoscopy (March 18).—The bladder and ureteric orifices were healthy. There was profuse bleeding from the right ureter; the urine from the left



FIG. 191.—The pelvis and the visible minor calices are of normal appearance, but the lowest group of minor calices is not seen.

ureter was free from blood. Intravenous indigo-carmin appeared from the left ureter in just under five minutes.

X-ray Examination (March 18).—A pyelogram of the right kidney (Fig. 191) showed a filling defect of the inferior calix major and related minor calices.

PRE-OPERATIVE DIAGNOSIS.—Tumour of the lower pole of the right kidney.

OPERATION (March 21—Mr. Roy Young).—The right kidney was exposed by

a loin incision. The organ was not enlarged. There was no external evidence of tumour, nor of any growth along the blood-vessels. Nephrectomy was performed, however, because of the dangerous hæmaturia and the abnormal pyelogram.

SUBSEQUENT PROGRESS.—Convalescence was uncomplicated. For five days after operation the amount of urine passed in twenty-four hours averaged about 20 oz. On the sixth day it increased to 54 oz., and thereafter the output was maintained at about this figure.

The kidney was bisected; one half, of which a drawing is shown (*Fig. 192*), was mounted as a museum specimen; the rest was utilized for histological examination.

MORBID ANATOMY.—The kidney is of normal size and shape. The large superior pyramid shows striate congestion, but otherwise the parenchyma is not obviously abnormal. A striking feature of the sectioned kidney is a circular mass of blood-clot, distending and firmly adherent to one of the uppermost of the lower group of minor calices, compressing an adjacent minor calix and also the opening of the



FIG. 192.—The bisected kidney. The angioma, semilunar in section, is seen at A. The opening of the inferior calix major is obstructed by the presence in an adjacent minor calix of a spherical mass of blood-clot, C. There is extensive hæmorrhage in the wall of the pelvis and several minor calices.



FIG. 193.—The angioma. The growth is seen to occupy the wall of the minor calix and to extend for a short distance along the interlobular septum. ($\times 7$.)

inferior calix major, thereby producing the filling defect seen in the pyelogram. Directly above this mass of clot, in the wall of the pelvis at the opening of the superior major calix, is an area of tissue of cavernous appearance, semilunar in section, but extending round the lower wall of the superior calix major, and in the adjacent minor calices of the inferior group, as far as the angle of deviation. This tissue constitutes the angioma. Much hæmorrhage is present in the inner layers of the pelvis, the inferior calix major, and the majority of the minor calices near the angle of deviation. The tissues of the hilum contain much blood, and the gross features suggest the presence of multiple foci of angiomatous tissue, but, as described below, these appearances are due to widespread hæmorrhages.

Microscopic Examination.—The low-power photomicrograph (Fig. 193) shows clearly the situation of the angioma in the renal pelvis. The portion represented is the wall of one of the minor calices in the region of the angle of deviation. Angiomatous tissue is present in the wall of the calix, and extends for a short distance into the kidney substance along the interlobular septum.



FIG. 194.—A portion of the angioma. Thin-walled blood-spaces of varying size, lined by flat endothelium and separated by septa of delicate fibrous tissue. Fairly numerous eosinophil leucocytes are present in the stroma. ($\times 130$.)



FIG. 195.—A portion of the angioma. Note the thin-walled blood-spaces and absence of hyperplasia of the endothelium. ($\times 50$.)

The cavernous tissue (Figs. 194, 195) consists of wide blood-spaces, lined by flattened endothelium, nowhere hyperplastic, and separated by fibrous septa, some very delicate, others thicker and containing a few smooth-muscle fibres. Numerous eosinophil leucocytes are present. In certain areas thrombosis has occurred and organization is in progress. Several of the blood-spaces near the angle of deviation which abut upon the renal pelvis have ruptured, and free communication has been established between the sinuses of the angioma and the minor calix, which is filled with blood-clot (Fig. 196). This lesion is the source of the hæmaturia.

The cavernous tissue is separated from the renal parenchyma by an area of young fibrous tissue which contains numerous capillary

blood-vessels lined by plump endothelial cells and is overrun by eosinophil leucocytes. No transition is found between the vessels of embryonic type and the wide spaces of the angioma. The tissue separating the cavernous spaces from the renal parenchyma is thus a sort of young granulation tissue, which for some

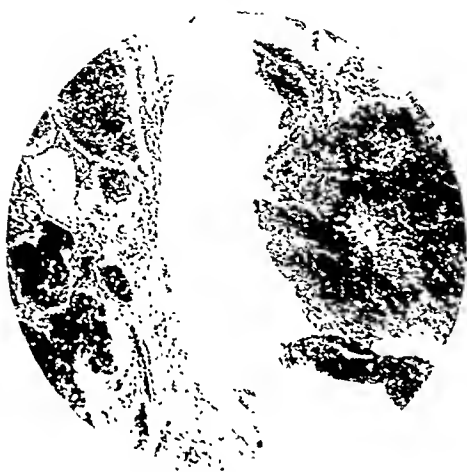


FIG. 196.—The lesion determining hæmat-
uria. Certain of the large blood-spaces have
ruptured and communicate freely with the
pelvis, which is filled with blood-clot. The
remains of the walls of the spaces are seen
ending freely in the cavity of the calix.
($\times 25$.)



FIG. 197.—The inflamed papilla. The
intertubular vessels are extremely engorged.
Many of the tubules have lost their epi-
thelial lining, while others show swelling
and regenerative changes in the epithelium.
($\times 50$.)

distance extends along the septa accompanying the interlobular vessels into the renal substance. There is some catarrh in the adjacent tubules and glomeruli, and casts or blood-corpuscles are present in the lumina of the tubules.

The pyramid entering the calix which is distended by blood-clot shows recent acute inflammatory change. There is some necrosis at the tip, leucocytic emigration, extreme engorgement of the intertubular veins, and desquamation of the tubular epithelium. At their termination the ducts of Bellini are devoid of epithelium and contain masses of granular debris and leucocytes, while at higher levels reparative processes with active proliferation of epithelium are in progress (Fig. 197).

The pelvic wall is œdematous and shows extensive subepithelial hæmorrhage (Fig. 198). A few large thin-walled vessels can still be distinguished, and these show margination

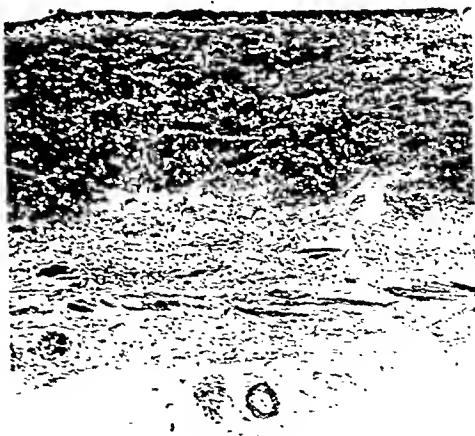


FIG. 198.—The wall of the renal pelvis. There
is very extensive subepithelial hæmorrhage. ($\times 20$.)

of the leucocytes, while at places there is marked perivascular infiltration of lymphocytes and mononuclear cells.

As mentioned previously, the fibro-fatty tissue of the hilum filling the spaces between the minor calices contained much blood, and the appearances suggested that there might be many scattered foci of angiomatous tissue in these situations. Microscopic examination, however, reveals no such structure, and the appearances are due solely to the presence of numerous small hæmorrhages.

With the exception of the papilla entering that calix which is filled by blood-clot, and the tubules and glomeruli close to the granulation tissue extending from the angioma into the kidney substance, the renal parenchyma is healthy.

DISCUSSION.

The morbid appearances described are of considerable complexity. The kidney shows three lesions, each of which may be associated with hæmaturia: (1) An angioma of the renal pelvis; (2) Extensive hæmorrhage in the sub-epithelial tissue of the renal pelvis; (3) A condition of papillitis with extreme vascular engorgement in the renal pyramid entering the calix, which is filled with blood-clot.

With regard to the second and third lesions, Quinby (1920) has stated that subepithelial pelvic hæmorrhage, together with a degree of congestion of the renal pyramids, may be the sole lesion present in so-called 'essential hæmaturia', as in two cases which he published. This has been the significant finding in eight similar cases which have come under the present author's notice. In the case now reported, however, the angioma of the pelvis is clearly the primary lesion. The subepithelial hæmorrhage has spread from the neighbourhood of the tumour, and the condition of papillitis has been determined by the presence in the related calix of a mass of clot derived from hæmorrhage from the ruptured angioma and firmly attached to it.

The primary lesion is a cavernous structure composed of thin-walled blood-spaces lined by flattened endothelium in no wise hyperplastic. Its margins show no evidence of infiltration or spread of a neoplastic process. The microscopic findings resemble those frequently met with in cavernous angiomata of the liver.

The origin and exact nature of angiomata is obscure. Some authors (Borst, 1923, et al.) apply to them the term 'hamartoma', used by Albrecht (1904) to designate neoplasms arising in developmental error and showing "lack of proliferation of their cells, absence of tendency to expansion or destructive infiltration, and the maintenance of the function of the constituent cells." Borst suggests that the mechanism of production of angiomata lies in local failure of development of the proper tissue—of skin, liver, kidney, etc.—with compensatory overgrowth of vascular mesenchyme.

The cutaneous naevi, being of course the most accessible, have been the most completely studied. Practically always congenital, and frequently multiple, they occur particularly in the region of the embryonal fissures, where rests of superfluous embryonic tissue are most likely to occur. Angiomata, however, show all stages of differentiation. The cirroid aneurysm is a simple overgrowth of a segment of the arterial tree, approaching closely in structure to normal adult blood-vessels. The ordinary congenital nevus shows a degree of anaplasia toward the embryonic type of blood-vessel and

a certain capacity for progressive growth and local malignancy. Finally, rare cases of metastasizing angiomata are on record in which death was caused by systemic dissemination of a cellular tumour composed of endothelial cells forming irregular capillary spaces (Ewing, 1918). Borst quotes from Kahle the occurrence in malignant angiomata of hæmopoiesis, a return to embryonic activity.

While many angiomata come under observation at a non-progressive stage, yet a period of growth in post-uterine life must often have preceded. Capillary nævi frequently enlarge, and they may also alter in type, showing cavernous transformation centrally, while growth may continue for a variable period in the peripheral zone of sprouting capillary buds. As Virchow (1867) pointed out, such changes may occur early, or they may arise late in life in capillary nævi long quiescent.

It is reasonable to assume that visceral angiomata may follow a similar course and that a tumour in such a situation as the kidney may therefore produce symptoms early or after a long period of latency. On the other hand, a tumour originally of cellular capillary type may become cavernous in structure, may develop a capsule, and ultimately lose all signs of active growth.

The term 'hamartoma' as applied to these tumours is of value in emphasizing, with regard to their genesis, the greater importance of developmental error than of extraneous stimulus. Precisely as in other tissues, however, the neoplastic lesions of blood-vessels form a continuous series from the most benign to the most malignant, and it does not appear to be of value to segregate the small group which conforms completely with Albrecht's definition.

The further point remains as to the possible relationship between such vascular tumours and chronic inflammatory processes. Ewing has stated that a definite relation between chronic granulation tissue and any of the characteristic vascular tumours can rarely be established. In the present case there is around the tumour a zone of tissue the seat of mild chronic inflammation and showing new formation of blood-vessels. It is probable that this represents merely a reaction of the surrounding tissues to the tumour mass.

REVIEW OF THE LITERATURE.

Virchow states that, with the exception of the liver, the kidney is the organ most frequently the seat of angioma, and that the latter is of not uncommon occurrence; but it is difficult to obtain confirmatory evidence of this from the writings of others. Thus Fukuda (1911) describes two angiomata of the renal parenchyma discovered accidentally at autopsy. He regards them as of great rarity. Kaufmann (1922), without giving details, states that they are rare, and Professor Muir informs me that he can recall no example of renal angioma found post mortem at the Western Infirmary. It must be admitted, however, that without purposeful search and microscopic examination small angiomata might be overlooked, or mistaken for foci of hæmorrhage.

From the literature 17 cases of renal angioma have been collected, but

all of these caused bleeding sufficiently severe to necessitate surgical treatment. They have been classified in order to determine whether any constantly recurring clinical feature might be of service in the differential diagnosis of cases of hæmaturia.

1. **Angiomata of the Renal Pelvic Wall.**—Six additional examples of angioma of the renal pelvis resembling that described have been found in the literature (*Table I*). The symptoms are very variable. Hæmaturia may be

Table I.—ANGIOMATA ARISING IN THE WALL OF THE RENAL PELVIS.

AUTHOR	AGE	SEX	SIDE	SYMPTOMS	NATURE OF LESION
Swan (1921)	19	M.	L.	Profuse hæmaturia for 3 days after football. Urgent	Cavernous angioma close to pelvis
Kidd (1924)	19	M.	R.	Attacks of hæmaturia with fainting, retention, etc., 10 years. Finally urgent	Dilated sinus-like spaces below epithelium of calix
Suter (1902) from Lutz (1925)	32	F.	—	Progressive hæmaturia 1½ years	Multiple pelvic angiomata
Jenkins and Drennan (1928)	33	F.	L.	Two previous attacks in 8 years; severe hæmaturia 1 week	Closely packed thin-walled vessels under epithelium of calix
Wheeler (1924)	37	M.	L.	Severe uncontrollable renal hæmorrhage without previous warning. No pain or tenderness	Angiomatous formation under epithelium of 3rd uppermost calix. Also a cluster of vessels under capsule
Author's case	61	F.	R.	Pain and severe hæmaturia 8 days. Urgent	Angioma of wall of pelvis
Lutz (1925)	62	M.	L.	Pain and hæmaturia 5 weeks	Loose cavernous tissue immediately under epithelium of calix

so severe and dramatic in its onset that immediate nephrectomy may be necessary to save life, or less severe attacks of hæmaturia may occur over a period of years. But it is fairly clearly brought out that in most cases the symptoms of a lesion of this type tend to run an acute course and rapidly to reach a stage when intervention of some sort is imperative.

The case of Jenkins and Drennan (1928) showed, in relation to an upper calix, an angiomatous growth one inch in diameter which had produced in an adjacent pyramid—apparently by pressure—necrosis, hæmorrhage, and desquamation, changes closely resembling those in the case submitted. These authors describe a mottled appearance in the pyclogram, and they compare this with the mottled appearance described by Jacobs and Rosenberg (1927), as is mentioned later. They suggest that this sign may be of diagnostic importance. In Swan's case (1921) hæmaturia originated after a game of football, and was so severe as to necessitate nephrectomy after three days.

Lutz (1925) describes the case of a male subject of 62 years who suffered from severe hæmaturia of five weeks' duration due to a pelvic angioma. He discusses the classification and the pathogenesis of such lesions. He regards all as congenital abnormalities due to local failure of development of the renal rudiment and ingrowth of excess of vascular mesenchyme. This may form a progressive tumour or it may be latent until the later years of life, when degenerative changes and increased blood-pressure, causing general or local passive congestion, may lead secondarily to widening of the vascular spaces so as ultimately to produce symptoms. In support of the congenital origin of these tumours he also quotes the case of Suter (1902) in which hypoplasia of the kidney and pelvis was associated with multiple pelvic angiomata. Wheeler (1924) illustrates the dramatic suddenness with which dangerous hæmorrhage may occur apart from predisposing trauma and without pain or tenderness or previous warning hæmorrhage.

2. Angiomata of the Renal Cortex.—Virehow states that angiomata of the kidney occur most frequently immediately under the capsule and that they may be multiple. From the literature only four such cases giving rise to symptoms have been collected (*Table II*). In these cases the tumour had attained to a relatively large size, yet in none did the bleeding constitute a surgical emergency.

Table II.—ANGIOMATA OF THE RENAL CORTX.

AUTHOR	AGE	SEX	SIDE	SYMPTOMS	NATURE OF LESION
Gile (1929)	37	M.	R.	Hæmaturia 9 months ..	Cavernous tumour (6 × 6 in.)
Judd and Simon (1928)	57	F.	L.	Hæmaturia 20 years ..	Tumour upper pole (1.5 × 2.5 cm.)
Deansley, quoted by Swan (1921)	66	M.	L.	Hæmaturia 6 months ..	Multiple capillary angiomata of cortex. One removed—recovery
McKenzie and Parkin (1929)	—	—	L.	Hæmaturia 2 weeks ..	Large angioma, upper pole

3. Angiomata of the Renal Pyramids.—The remaining 7 cases consist of angiomata of the pyramids, and of these full information is available in 6 (*Table III*). The subjects range in age from 23 to 54. In none was emergency surgery necessary.

The case of Jacobs and Rosenberg (1927) is inserted in this series with some hesitation. These authors describe the lesion which they report as a telangiectasis or hæmangioma simplex, involving the upper pole of a kidney, particularly certain pyramids and the wall of the pelvis, and they differentiate it sharply from the cavernous hæmangiomata published by other writers. In doing so they imply that the other recorded cases exhibit histological evidence of potentiality for progressive neoplasia. A survey of the literature, however,

does not convey the impression that the majority of vascular tumours of the kidney show hyperplastic endothelium or evidence of progressive growth. The reasons which led to the adoption of the title 'cavernous hæmangioma' in our case might justifiably support the inclusion of this last in the same group. Jacobs and Rosenberg describe a mottled appearance seen in the pyelogram, and suggest that it may be due to entry into the blood-spaces of the lesion of the pyelographic shadow-producing medium, and that this appearance may be of some diagnostic value.

Table III.—ANGIOMATA OF THE PYRAMIDS.

AUTHOR	AGE	SEX	SIDE	SYMPTOMS	NATURE OF LESION
Morris (1901)	23	F.	L.	Severe hæmaturia and pain in loins for some years	Multiple angiomata of pyramids
Stieda (1923)	29	M.	R.	6½ years' progressive hæmaturia	Angioma of pyramid with calcification of stroma
Simpson (1927)	30	F.	R.	18 months' progressive hæmaturia	Angioma size of pin-head at papilla-tip
Begg (1926)	33	M.	R.	Hæmaturia 2 years previously after strain; recurring recently after another strain	Blood cyst of lower pole originating in angioma of a lower pyramid, and cutting off lower calix major
Jacobs and Rosenberg (1927)	36	F.	L.	Backache and hæmaturia 4 weeks. Mottling in pyelogram	Extensive telangiectasis of an upper pole and also of the renal pelvis
Newman (1915)	54	M.	L.	Hæmaturia 4 years ..	Angioma of uppermost pyramid. Encapsulated
Rousing (1918) from Lutz (1923)	—	—	—	Sharp bleeding	Angioma size of pea replacing renal papilla

Begg (1926) states that hæmaturia was twice precipitated by a 'strain' in his case, which showed a blood cyst, 11 cm. in diameter, of the lower pole of the right kidney, arising apparently in a cavernous angioma and communicating with the pelvis. Morris (1901) describes multiple angiomata in a male subject who died of septicæmia, after having survived many attacks of hæmaturia. Stieda (1923) quotes a case in which the external appearance of the kidney was perfectly normal, and at the first operation decapsulation only was performed. Hæmorrhage continued, and nephrectomy was necessary later. The kidney contained an angioma of a pyramid. He discusses the causal inter-relation between calcification of the papillary stroma and the angiomatous formation, and also the connection of exposure to cold and over-strain with the occurrence of hæmaturia.

FACTORS DETERMINING HÆMATURIA FROM RENAL ANGIOMATA.

The total number of recorded cases is too small, and the individual details are too diverse, to permit the drawing of wide conclusions. The majority of the cases have been discovered through severe hæmaturia necessitating surgical intervention. Renal angiomas appear to have an especial tendency, not shared by angiomas in other situations, to give rise to hæmorrhage. Three factors may contribute to this property: (1) The situation and structure of the tumour; (2) The liability of the tumour to sustain injury; (3) The possibility of obstruction of the venous return from the tumour.

Tumours of the renal pelvis or pyramids are in direct relationship with the pelvis, while those of the renal cortex, at first latent, may grow until they also abut upon the pelvis. The tumour is generally of cavernous type, and the thin-walled blood-spaces covered by only a thin layer of soft pelvic or renal tissue are relatively easily damaged. It is clear how severe trauma or strain, as in the cases of Begg and Swan, may lead to hæmorrhage from such a tumour, but even in the absence of such definite trauma the anatomical structure and relations of the kidney and its physiological excursion during respiration may be factors of importance. The kidney is not firmly anchored to the posterior abdominal wall, but lies in a loose fibro-fatty capsule which allows it to move with the respiratory excursions of the diaphragm and with movements of the trunk. While angiomas of the renal parenchyma are protected, these movements must impose a certain shearing stress upon tumours of the pelvis. Furthermore, the pelvic wall is continually contracting and relaxing as it expels the accumulating urine, and this also will subject the delicate tumours to strain. Finally, if hæmorrhage should supervene, the blood is rapidly washed away by the urine and clotting is not permitted.

Passive congestion also appears to be an important etiological factor, and it has received much attention in the literature. The kidney, as described above, is mobile within limits, yet its vessels are short, straight, and firmly anchored at their origins, and the veins join the inferior vena cava at right angles. Newman (1907) states that the vascular torsion occurring in a preternaturally mobile but otherwise healthy kidney may lead to severe hæmaturia. Hence we may suppose that in the presence of an angioma a less extreme degree of mobility may precipitate hæmorrhage. Lutz emphasizes the importance of passive congestion in producing engorgement, enlargement, and finally rupture of a congenital angioma long latent.

Newman (1914) collected a series of thirteen cases of varix of the renal papilla. These were not apparently true angiomas, but engorgements of the pre-existing veins of the papilla, with a tendency to hæmorrhage. He suggests that vascular obstruction at the boundary zone is the primary cause of the congestion, and quotes good results from nephrotomy, down to the boundary zone, performed roughly in order to produce the maximum of damage to the vascular arches. He states that this treatment diminishes the arterial supply to the papillæ and so relieves the engorged venules. Payne and MacNider (1916), in a series of cases of unilateral symptomless hæmaturia, found that areas of chronic nephritis with fibrosis, occurring at the bases of the pyramids

and at the boundary zone, had caused varicosity and rupture of the veins of the related pyramids. They succeeded in producing similar lesions experimentally in dogs. Extensive nephrotomy relieved symptoms in their eleven human cases. These authors regard nephrotomy as a method of treatment of choice, since it will reveal the presence of other more serious lesions, and in the absence of the latter will lead to cure in a large proportion of cases. Of Newman's cases, complete information is available in twelve. Of these, the varix was in the left kidney in nine. This is suggestive, in view of the longer course of the left renal vein, which also crosses the front of the aorta and might therefore be expected to suffer more frequently from obstruction than the right.

A combination of the three factors described may endow angioma of the renal pelvis with its especial tendency to sudden severe hæmorrhage.

DIAGNOSIS.

The diagnosis of renal angioma is difficult, and the rarity of the lesion is a further handicap. In the past the diagnosis has generally been made by the pathologist. Careful clinical investigation and exclusion of other causes of hæmaturia may establish a state of reasonable probability, but more we can hardly expect.

Simple rest in bed will relieve and therefore eliminate hæmorrhage due to congestion of a prolapsed preternaturally mobile kidney, but it will tend also to alleviate bleeding from other more serious conditions. The urine should be investigated for the presence of tubercle bacilli or other organisms and for the presence of pus cells and casts. Negative findings will help to exclude tubercle and other infections, and the absence of casts will lessen the probability of hæmorrhagic nephritis. The exclusion of this last will be supported if the complaints of the patient and cystoscopic examination demonstrate unilateral renal hæmaturia. X-ray examination will yield negative more often than positive information, and it must be in rare cases only that a diagnostic picture such as that described by Jacobs and Rosenberg and Jenkins and Drennan will be obtained. In fact it seems more probable that the presence of blood-clot in the renal pelvis may produce a filling defect and support an error of diagnosis, as in our case, in which the pyelographic appearances suggest pressure by a tumour upon the lower group of calices. Nevertheless we may exclude renal stone, and it is improbable that a normal pyelogram will be obtained in the presence of one of the more gross renal tumours. Definite information will not be obtained from renal function estimations, but it seems probable that the functional capacity of the kidney in a case such as ours will be less impaired than it would be in a case of advanced tumour, stone, or tuberculosis.

It is of importance to exclude that type of essential hæmaturia arising in subepithelial pelvic hæmorrhage, as this not uncommon condition appears to respond to non-operative treatment, and any operation therefore must be considered unjustifiably mutilating unless other less drastic lines of treatment have been explored without success. Such measures as that recommended by Rytina (1920)—namely, the intrapelvic injection of 4 to 10 c.c. of a silver nitrate solution 1 to 5 per cent in strength—would appear to have some value

as a therapeutic test. This author reports the cure of three cases, one of which had been aggravated rather than improved by a previous nephrotomy.

The clinical course of the illness acquires more definite significance after such extensive investigation is complete. In the absence of features indicating another lesion, it might be justifiable to suspect the presence of an angioma of the renal pelvis when hæmaturia is met with, particularly in a fairly young person, dramatic in onset, and so severe as imperatively to demand surgical intervention. The acuity of the course which is generally followed by the disease is illustrated in *Table I*.

TREATMENT.

The angioma is a simple tumour, generally fairly small in size, and sacrifice of an entire kidney may appear to be a surgical extravagance. Superficial angiomas of the parenchyma probably rarely require surgical treatment. Tumours or varices of the papillæ may be cured by papillectomy or nephrotomy. If nephrotomy revealed in an accessible position a small angioma, it would be attractive to attempt its removal by some form of electro-coagulation. But in a case such as that reported above, in which hæmorrhage so severe as to endanger life occurred in eight days, a possibly unsuccessful operation is not lightly to be undertaken, and conservation would be difficult and undoubtedly hazardous in such a case where a fairly extensive angioma was present in a somewhat inaccessible situation. In these circumstances, and in the presence of another kidney free from disease, nephrectomy would seem to be the swiftest and surest road to a satisfactory cure.

SUMMARY.

1. A case is reported in which dangerous hæmaturia arose from a cavernous hæmangioma of the renal pelvis.
2. Seventeen examples of hæmangioma of the kidney have been collected from the literature.
3. The material has been classified and an attempt has been made to correlate symptoms with the situation and pathological characters of the lesions.
4. The treatment of the various lesions is briefly considered.

I am much indebted to Mr. Roy Young, Visiting Surgeon to the Western Infirmary, Glasgow, for the use of his clinical records, the pyelogram, and the drawing by Mr. R. M. Buchanan, M.B., of the museum specimen. The excellent photomicrographs are by Mr. John Kirkpatrick, of the Department of Pathology, Glasgow University.

This work has been done under a grant from the Medical Research Council, for which I wish to express my thanks.

BIBLIOGRAPHY.

- ALBRECHT, H., *Zentralb. f. allg. Pathol. u. pathol. Anat.*, 1904, xv, 546.
BEGG, R. C., "Solitary Hæmorrhagic Cysts of Kidney", *Brit. Jour. Surg.*, 1925-6, xiii, 649.

- BORST, M., *Pathologische Anatomie* (Aschoff), 1923, i, 687. Jena.
- DEANSLEY, quoted by SWAN, in *Proc. Roy. Soc. Med. (Urol. Sect.)*, 1921, xiv, 35.
- EWING, J., *Neoplastic Diseases*, 1928, 240. Philadelphia.
- FENWICK, E. H., *Clinical Cystoscopy*, 1904, 392. London.
- FUKUDA, quoted by STIEDA (1923), *Verhandl. Japan. pathol. Gesellsch.*, 1911 (5 and 6, iv), 564.
- GILE, H. H., "Hæmangioma of the Kidney", *Surg. Gynecol. and Obst.*, 1929, xlviii, 555.
- JACOBS, P. A., and ROSENBERG, W., "Telangiectasis of Kidney Simulating Renal Tumour", *Jour. of Urol.*, 1927, xvii, 337.
- JENKINS, J. A., and DRENNAN, A. M., "Cavernous Hæmangioma of the Kidney", *Ibid.*, 1928, xx, 97.
- JUDD, E. S., and SIMON, H. E., "Angioma of Kidney", *Surg. Gynecol. and Obst.*, 1928, xvi, 711.
- KAUFMANN, E., *Spezielle pathologische Anatomie*, 1922, ii, 1091. Berlin.
- KIDD, F., "Cavernous Angioma of Kidney", *Proc. Roy. Soc. Med. (Urol. Sect.)*, 1924, xvii, 56.
- LUTZ, G., "Profuse Hæmaturie infolge eines Nierenbeckenkavernoms", *Zeits. f. urol. Chir.*, 1924-5, xvii, 93.
- MACKENZIE, D. W., and PARKIN, G. A., "Renal Tumours", *Canad. Med. Assoc. Jour.*, 1929, xx, 616.
- MORRIS, H., *Surgical Diseases of Kidney and Ureter*, 1901, i, 630. London.
- NEWMAN, D., *Movable Kidney*, 1907, 40. London.
- NEWMAN, D., "Renal Varix and Hyperæmia as Causes of Symptomless Renal Hæmaturia", *Brit. Jour. Surg.*, 1913-14, i, 4.
- NEWMAN, D., "Symptomless Renal Hæmaturia arising from Tumours, Aneurysms in the Renal Pelvis, Calculus, and Early Tuberculosis", *Ibid.*, 1914-15, ii, 554.
- PAYNE, R. L., jun., and MACNIDER, W. B., "The Surgical Problem of Unilateral Symptomless Hæmaturia", *Jour. Amer. Med. Assoc.*, 1916, lxvii, 918.
- QUINBY, W. C., "The Pathology of the Renal Pelvis in Two Cases showing Hæmaturia of the So-called 'Essential' Type", *Jour. of Urol.*, 1920, iv, 209.
- ROUSING, quoted by LUTZ (1925), *Hülstleinwils Lehrbuch der Chirurgie*, 1918, ii, 382. Jena.
- RYTINA, A. G., "Treatment of Essential Renal Hæmaturia by Intrapelvic Injections of Silver Nitrate", *Jour. of Urol.*, 1920, iv, 317.
- SIMPSON, G., "Angioma of the Kidney", *Proc. Roy. Soc. Med. (Urol. Sect.)*, 1926-7, xx, 728.
- STIEDA, A., "Angiom einer Nierenpapillenspitze als Ursache schwerster Blutung", *Beitr. z. pathol. Anat.*, 1923, lxxi, 545.
- SUTER, quoted by LUTZ (1925), *Zbl. f. Harn. u. Sexualorg.*, 1902, 27.
- SWAN, R. H. J., "Angioma of the Kidney", *Proc. Roy. Soc. Med. (Urol. Sect.)*, 1920-1, xiv, 35.
- VIRCHOW, R., *Die krankhaften Geschwulste*, 1867, i, 306. Berlin.
- WHEELER, WM., "Some Renal Tumours", *Surg. Gynecol. and Obst.*, 1924, xxxviii, 143.

ERRATA

Under the article HÆMANGIOMA OF THE KIDNEY in our October number

Page 320 tenth line from top for DRENNAN read DRENNAN,
and thirteenth line from bottom for ROUSING read
ROUSING.

SARCOMA AND TRAUMA.

By R. J. WILLAN, M.V.O.,

SURGEON TO THE ROYAL VICTORIA INFIRMARY, NEWCASTLE-UPON-TYNE.

It has been established in a court of law that for purpose of compensation sarcoma can be held to be a sequel to trauma. The following case illustrating this possible relationship is of considerable interest. The accident was officially reported to the patient's employers, and he continued to do



FIG. 199.—Skiagram of hip-joint taken three months after the accident.

arduous work for ten days after the accident, when his employment ceased. I saw him first exactly three months after the accident, when he complained of acute but vague pains. At that time the physical signs were normal, as were radiograms of the hip-joint and femur.

A spontaneous fracture of the neck of the right femur appeared within

eight months after the accident, the exact date of the fracture being uncertain; at the same time deposits of growth were found in the clavicle and humerus. The patient died from cachexia one year and eleven months after the accident, and a post-mortem examination confirmed the ante-mortem diagnosis of sarcoma. The possibilities are: (1) Primary growth in the femur, with secondary deposits in the clavicle and humerus; (2) Primary growth in the clavicle or humerus and secondary deposits in the femur, for no radiogram of the shoulder area was taken until eight months after the accident; (3) That the accident was a coincidence and in no way connected with the sarcoma.



FIG. 200.—Skiagram of hip-joint taken eight months after the accident.

Assuming that the accident really was responsible for the onset of sarcoma, it is interesting to compare the two radiograms—*Fig. 199*, taken three months after the accident, and *Fig. 200*, taken eight months after. It is also interesting to speculate whether the sarcoma arose from the traumatized tissue, and if so, when the sarcomatous change took place.

CASE REPORT.

HISTORY.—On July 25, 1928, Thomas B., age 51, labourer, was working in a railway truck and sustained an accident. The 'Accident Report', which he filled in for compensation purposes, stated that he had sustained a "bruise

to the thigh" caused by "slipping and falling to the ground when climbing out of the truck". The accident was not reported officially until fourteen days after its occurrence, but liability for compensation was accepted and was paid. Evidence was subsequently obtained as to the presence of the bruise over the right femoral trochanter, also that the bruise lasted for about six weeks.

The man continued to work as a labourer until Aug. 4—that is, ten days after the accident—when he was paid off owing to slackness of trade. Prior to the accident his panel doctor, Dr. MacElroy, of Blackhill, states that he was quite well in every way.



FIG. 201.—Skiagram showing the growths in the outer end of the clavicle and the upper end of the shaft of the right humerus.

FIRST EXAMINATION.—The patient continued to have unaccountable pains which simulated sciatica and lumbago, so in October, 1928, Dr. MacElroy sent him to see me at the Royal Victoria Infirmary, Newcastle, as an out-patient. He then had no abnormal physical signs in the right hip and lumbar vertebral joint areas—that is, there was no muscle wasting or rigidity, nor was there any limitation of movement in the right hip-joint or lumbar vertebral joints. His hip-joint was X-rayed on Oct. 25 (*Fig. 199*), but nothing abnormal could be made out. A radiogram of the right side of the pelvis also showed normal shadows. No complaint whatever was made of the shoulder-joint, either to

Dr. MacElroy or to myself; it was therefore not examined by me on this occasion. The pains gradually became worse, the hip-joint area swelled up, walking became increasingly more difficult, and by January, 1929, the patient was bedfast.

SECOND EXAMINATION.—I next saw him in March, when there was a pathological fracture of the neck of the right femur. He then complained of pain in the shoulder for the first time, and it was noticed that he had a swelling in the region of his right shoulder and at the outer end of the right clavicle. X rays (*Figs. 200, 201*) showed a growth in the neck of the femur, the outer end of the clavicle, and the upper end of the shaft of the right humerus.

ON ADMISSION.—In October the patient was admitted under my care at the Royal Victoria Infirmary, Newcastle, as an in-patient; radiograms showed that the growths had increased in size. His general condition became

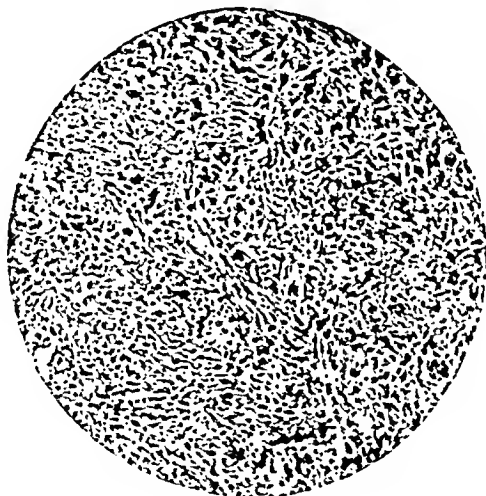


FIG. 202.—Microphotograph of growth in right femur. ($\times 80$.)

progressively worse, both right hip-joint and right shoulder area swellings gradually increased in size, and he died from cachexia on June 10, 1930—nearly two years after the accident.

AUTOPSY.—A post-mortem examination made by Dr. W. E. Hume and myself revealed a growth of large size originating in the neck of the right femur with a fracture; the growth was like brain in appearance, and similar growth distended the outer half of the right clavicle and upper end of the right humerus; the growth had the appearance of a sarcoma. There was no evidence of neoplasm in the thoracic or abdominal viscera.

PATHOLOGICAL REPORT.—A piece of the growth was submitted to Professor Stuart MacDonald for examination, and he reports: "This tumour is certainly neoplastic, and is of the nature of a spindle-cell sarcoma. This portion of tissue is well preserved, is not necrotic, and shows no evidence of bone formation." *Fig. 202* is a microphotograph of the growth.

SUMMARY.

The case is a spindle-celled sarcoma of the femur associated with an undoubted injury to the same area. X rays showed a normal bone three months after the accident; within the next five months there was a spontaneous fracture from the growth. Similar growths were found in two other bones. Death took place one year and eleven months after the accident. Sarcoma was confirmed by a post-mortem examination. It is true that the connection between the trauma and sarcoma rests only on circumstantial evidence, but it appears to me unlikely that a more certain chain of evidence can be obtained than in this case.

I am indebted to the Honorary Physician to the Electrical Department of the Royal Victoria Infirmary, Newcastle, for *Fig. 199*, to Dr. W. D. Arnison for *Figs. 200 and 201*, and to Professor Stuart MacDonald for the pathological report and the microphotograph.

FATAL CARDIAC FAILURE OCCURRING IN PERSONS WITH ANGULAR DEFORMITY OF THE SPINE.

By CAREY F. COOMBS,

PHYSICIAN, AND IN CHARGE OF UNIVERSITY CENTRE OF CARDIAC RESEARCH,
BRISTOL GENERAL HOSPITAL.

DURING the past few years I have seen four examples of the condition described in the title of this paper. Brief notes of these follow:—

Case 1.—A married woman, age 38, was admitted under my care at the Bristol General Hospital suffering from dyspnoea of twelve months' duration. For the first three or four months this had been worse, and dropsy had been added to it. There was pronounced cyanosis of the face, and the fingers were clubbed and purplish. The pulse-rate was 130 per minute, the rhythm being regular. The blood-pressure was 100 mm. Hg systolic, 80 diastolic. There was marked kyphosis of the thoracic vertebrae, with compensatory lordosis in the lumbar region, said to date from the age of 2 years. The cardiac impulse was increased in area towards the left, and was diffuse. A cantering rhythm was heard at the apex, and the pulmonic second sound was much accentuated. The oedema was of the cardiac type, being limited to dependent parts, but it was extreme and had to be relieved by the insertion of Southey's tubes into the legs. The urine contained albumin and some blood. The patient was temporarily relieved by treatment, but became strange in her manner and developed bronchitis, dying twelve days after admission.

Autopsy.—Post mortem the principal change was a globular dilatation of both ventricles, without valvular or pericardial disease. The walls of the two ventricles were approximately of the same thickness.

Case 2.—A youth of 19 was admitted under me at the General Hospital for abdominal pain with vomiting and fever. At the age of 7 he was operated on for tuberculous of the hip, and by the time he was 13 had developed angular curvature of the cervico-dorsal spine, apparently due to vertebral caries. He had been under treatment for this since, and was recently considered to have tuberculous in both lungs. While he was in the hospital he passed through an illness which was labelled bronchopneumonia. The sputum contained pneumococci but no tubercle bacilli. He was cyanosed on admission, but while he was convalescent from this acute illness he became more so, and developed a general dropsy. The cardiac dullness became widened, a cantering rhythm was noted at the apex, and marked accentuation of the second sound at the pulmonic base. Below this, over a restricted area in the centre of the precordium, a systolic murmur was heard. The pulse-rate varied from 110 to 140, but the rhythm was normal and there was no alternation. The blood-pressure reading was 130/110. While the patient was in this state his parents insisted on taking him home. He died shortly after. There was no autopsy.

Case 3.—A man of 23, admitted to the General Hospital under my colleague, Professor E. W. Hey Groves, for spinal tuberculosis, developed symptoms of circulatory failure. The onset of the spinal lesion dated back to the age of 2, but he had been able to walk till six months before admission. There was a marked kyphosis, with its apex in the seventh dorsal vertebra, and a spastic paraplegia of the lower limbs. He was deeply cyanosed, and had been short of breath for about four months. The pulse-rate was 110 and its rhythm regular. The blood-pressure was 160/110. The back and legs had recently become oedematous. The cardiac dullness was

HEART FAILURE AND SPINAL DEFORMITY 327

increased to the right, and a triple rhythm was heard at the apex. The urine was albuminous. The patient became rapidly worse and died.

AUTOPSY.—Post mortem the thoracic aorta was found to follow the dorsal spine into an angle, at the apex of which it was sharply kinked and therefore narrowed. The lungs were fibrous, and adherent to the pulmonary artery at its base. Both ventricles showed considerable dilatation, and the wall of the right ventricle was relatively thickened.

Case 4.—A man of 29 was seen by me in consultation with Dr. R. B. Britton on account of rapidly ingravescent cardiac failure. There was a kypho-scoliosis of the dorsal spine which had been first noted at the age of 3. It was said that his brother had been similarly deformed, and that in his case also this had ended in cardiac failure. The patient was dyspnoic and cyanosed, and had been so for two months. Recently he had become dropsical, and was now œdematous to the waist. The pulse-rate was 120 and the rhythm regular. The blood-pressure was 120/100. The heart-sounds were weak, but the aortic second sound was much accentuated. A 'bruit de galop' was noted. There were râles at the bases, and a trace of albumin in the urine. He died a few days later. Post-mortem examination was not feasible.

COMMENT.

All these patients displayed a gross deformity of the dorsal spine. In three there was an angular kyphosis of tuberculous origin. In the other the angulation was lateral as well as antero-posterior, and was said to have occurred in another member of the family, in association with symptoms of cardiac failure like those which my patient experienced. All these four subjects had been deformed for many years; three of them, indeed, since early childhood; but not until a few months before death—twelve months in one case, less than six months in the others—were there any cardiac symptoms. In all, the first complaint was of dyspnoea, and this increased rapidly until it reached an extreme degree. By the time that I saw them, all four were very cyanosed, and in one case—that of the woman—the fingers were clubbed and purple. In all of them there was an extreme and increasing dropsy up to the waist-line.

The pulse was quick but regular in every instance; the blood-pressure was low and the pulse-pressure gap very narrow in three of the four patients. Unfortunately no electrocardiograms were taken, as all four patients were extremely ill when first seen. Alternation of the pulse was not detected in any of them.

The physical signs were so distorted by the deformity of the chest that not much could be definitely established. It is, however, worthy of note that the impulse was always diffuse and the basic second sound relatively loud, while in all a 'bruit de galop' was heard. Physical signs of bronchitis, amounting in one instance to a bronchopneumonia, were also observed.

Post-mortem examinations were made in two cases (*Cases 1 and 3*). These, while incomplete, did at least confirm and explain the evidences of cardiac embarrassment. In *Case 1*, that of the woman of 38, a globular enlargement of the heart was noted, in which both ventricles shared, the wall of the right chamber being as thick as that of the left. Neither pericardium nor valves showed any gross change. This is true also of the heart in *Case 3*. Here we noted a remarkably acute angulation of the aortic arch, which had

been thus penalized for clinging too faithfully to the deformed spine. In this case also there was some fibrosis round the root of the pulmonary artery, as well as a manifest reduction in the capacity of the lungs.

The references to this form of cardiac failure are almost limited to Continental journals. There are but few papers in English dealing with the subject. I shall not attempt a review of what has been written, but one or two points are of general interest. First, the aortic kink seen in one of my cases was described by Morgagni. Second, it is stated by some orthopædic surgeons that enlargement of the heart is noted in a majority of kyphotic patients. Finally, three influences have been blamed for the cardiac failure: aortic kinking, inadequate air-space in the lungs, and restriction of the diaphragmatic excursion. Whatever the explanation, it is clear that there is a real danger of untimely death from cardiac failure in the victims of angular deformity of the spine, and it is by way of affording to the orthopædic surgeon an added reason for attempting to prevent that deformity that this note has been written.

*SHORT NOTES OF
RARE OR OBSCURE CASES*

**PIGMENTATION FROM BISMUTH ABSORPTION AFTER
THE USE OF B.I.P.P.**

BY SIR W. I. DE COURCY WHEELER, DUBLIN.

BISMUTH in the treatment of syphilis was abandoned for a time some years ago owing to the severe toxic effects of the salts then used. Toxicity was found to be largely influenced by the rate at which the metal entered the circulation.



FIG. 203.—Chronic osteomyelitis of the femur before the operation.

The most notable effects of poisoning were stomatitis, nephritis, and enteritis. The commonest sign of intolerance is a slaty-blue line commencing on the gums



FIG. 204.—Chronic osteomyelitis of the femur after radical operation, showing B.I.P.P. *in situ*. (Note pathological fracture.)

before or behind the incisor teeth. This blue deposit extends to the entire gum margins or inner side of the cheeks from continuous absorption. In severe cases a condition of *cancrum oris* may be found. Before the War pigmentation of the gums was usually associated with lead poisoning or purpura, or the use of charcoal tooth-powders; during the War it was seen occasionally following the application of bismuth paste to open wounds.

The figures illustrate the case of a boy, age 15, who had suffered from chronic septic osteomyelitis and multiple sinuses of the femur for many years (*Fig. 203*). The bone was exposed through an incision from the trochanter to the knee and a radical operation performed with the aid of a motor saw. About a teaspoonful of B.I.P.P. was smeared over the raw surfaces (*Fig. 204*). About a week after operation the patient looked toxic and albumin had appeared in the urine. On examination of the mouth, the under surface of the tongue was found deeply pigmented. The lower alveolar margin both



FIG. 205.—Deep pigmentation (suggesting imminent sloughing) of the gums, under surface of the tongue and lower lip, and the mucous membrane of the cheeks, after the application of B.I.P.P. in a case of osteomyelitis.



FIG. 206.—Same case six weeks later after treatment by nineteen intravenous injections of ametoxy given on alternate days. Suction drainage was applied to the reopened wound.

front and back was in the same condition. The deep surface of the lower lip was also deeply stained, as was the inner side of each cheek (*Fig. 205*). The appearance was such as to suggest the possibility of sloughing. The characteristic translucency of the blue stains could be well seen by means of a Cameron light.

The patient was treated on alternate days by intravenous injections of ametoxy, a preparation used as an antidote to poisoning from N.A.B., but said to be equally effective in poisoning from other heavy metals. The wound in the thigh was laid completely open, and a large cigarette drain with a catheter in the middle was connected with an electrical aspirator (Lovac) under the bed. A large amount of bismuth-stained septic material was removed in this way. In about a fortnight the pigmentation was less marked and the patient's condition improved (*Fig. 206*). The operation was performed on Jan. 30, 1930. Six months later, after treatment in an open-air hospital, the pigmentation had almost disappeared.

A CASE OF CONGENITAL STENOSIS OF THE DUODENUM.

By LENNOX GORDON,

ASSISTANT SURGEON, SOMERSET HOSPITAL, CAPE TOWN.

WHILE stenosis of the duodenum of congenital origin is a well-recognized abnormality, its occurrence is rare. The following case is noted as it brings out some points of interest, the chief being perhaps the age to which the child lived— $2\frac{1}{2}$ years—in spite of a very marked degree of stenosis. The difficulty in diagnosis is also of interest.

The patient was a 'coloured' boy, age 2 years and 6 months, who was admitted to hospital with the query diagnosis of 'nephritis', this diagnosis evidently being suggested by the fact that the child had œdema of the face, hands, and legs.

HISTORY.—The mother was a coloured woman of poor mentality, and it was not until after the child's death and careful questioning that she gave the following important history. She stated that the patient had suffered from frequent attacks of vomiting, the abdomen became distended, but this distension subsided as the vomiting ceased. These attacks occurred about once a month, and during the quiet intervals the child, although always thin, seemed well and contented. The mother had consulted a doctor about the patient because of the swelling of the extremities and face, and the child was not taking its food well. There was no distension of the stomach at the time.

COURSE.—The patient was admitted to the pædiatric department of the hospital and was under medical observation. The œdema disappeared within a few days, and was thought to have been due to some form of dietary deficiency; there was no evidence of nephritis. The child was for three weeks under careful medical observation, and nothing definite could be made out beyond that he appeared ill nourished. There was no vomiting, and no distension of the abdomen was noted. He passed some worms, for which a vermifuge was given.

After being in hospital three weeks the child was seized late one night with sudden acute symptoms. He started vomiting, the abdomen became greatly distended, the temperature rose, and the pulse-rate increased. By next morning he was extremely ill, the abdomen was greatly distended, and the vomiting was frequent and had now become bile-stained. Everything pointed to an acute obstruction and surgical aid was asked for. The child was now so extremely ill that the chance of relieving any obstruction by operation appeared remote. As a forlorn hope a small laparotomy opening was made under local anæsthesia and light ether, the stomach having first been washed out and a large kidney dish of bile-stained stomach contents drawn off. On opening the abdomen a greatly distended stomach and first part of duodenum was found, but before any further investigation or operative procedure could be carried out the child became suddenly worse.

As one wished to get the patient off the operating table alive the abdomen was closed, but he died while being stitched up.

POST-MORTEM EXAMINATION (carried out by Professor Ryrie).—Beyond a stenosis of the duodenum no other pathological lesion was discovered.

The duodenum was constricted in its second part in the region of the ampulla of Vater. The length of the constricted portion was about three-quarters of an inch, and the stricture just admitted a probe 3 mm. in diameter. Both the stomach and duodenum proximal to the stricture were markedly dilated, the distended part of the duodenum being as large as the stomach; the muscular walls were hypertrophied and the pyloric opening was freely patent. The opening of the common bile-duct was involved in the stricture, being more to the distal side, but there was no apparent obstruction of the bile-duct, as bile passed freely into the proximal

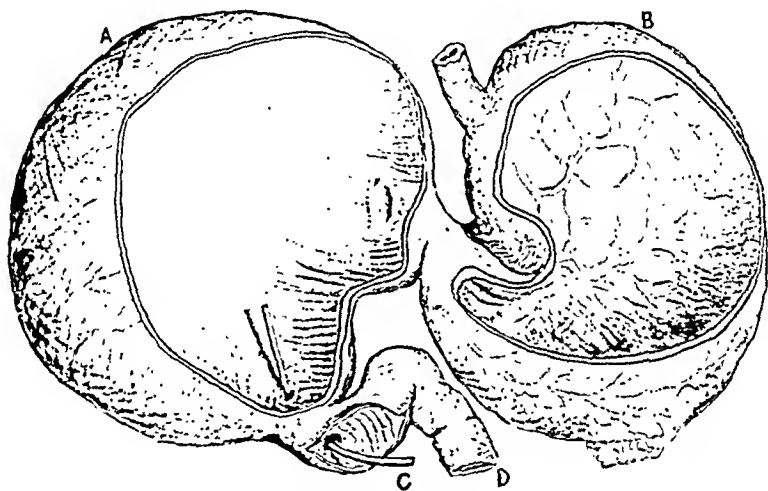


FIG. 207.—Appearance found on autopsy. A, Dilated duodenum; B, Stomach; C, Probe through stenosis; D, Duodenum. ($\times \frac{2}{3}$.)

dilated part of the duodenum. This would explain why the vomited material was bile-stained. The post-mortem appearance is shown in the accompanying sketch (*Fig. 207*).

Comment.—The special points of interest in this case appear to be: (1) The age to which the child lived, namely $2\frac{1}{2}$ years; (2) The fact that while under careful observation for several weeks there was no evidence of distension or obstruction.

These cases of duodenal obstruction usually die within the first few months of life. The longest case hitherto recorded is that of a girl who lived $2\frac{1}{2}$ years, and then died as the result of an acute obstruction from an impacted portion of food in the stenosed opening. The present case is therefore also interesting as regards the child's longevity.

The pathology has been discussed by various writers. The main feature

is that (apart from obstruction due to bands and adhesions) the stricture occurs either in the region of the papilla of Vater or at the duodenojejunal junction, the site being determined by Bland-Sutton's generalization that these occlusions occur at the site of embryological events. At the region of the papilla of Vater the liver and pancreas bud off, and this region is the meeting-place of the fore- and mid-gut.

The point of practical interest is the diagnosis. If the symptoms are recognized early, operation may save the patient's life. The chief symptom is vomiting; this starts usually soon after birth; it is in the large majority of cases bile-stained, and is forcible in character. The child is constipated, and wasting is evident. The symptoms closely resemble those of congenital pyloric stenosis. The points which may help to distinguish the two are that in duodenal stenosis the vomiting is said to begin at birth, it is usually bile-stained, and, further, no tumour is to be felt. An X-ray picture will of course be of great value, showing that food passes the pylorus and that it is the duodenum which is distended.

The treatment is surgical. The child's life may be saved by a short-circuiting operation, or, if the obstruction is due to bands, by dividing these.

A CASE OF CONGENITAL ATRESIA OF THE SMALL INTESTINE.

By CYRIL POLSON,

LECTURER IN PATHOLOGY, UNIVERSITY OF LEEDS.

THE present case is an example of atresia of the ileum, and resembles that described by Pasley¹ in 1929. It belongs to the third group of Forssner's² classification—namely, the type in which there is a complete break in the continuity of the small intestine, with the closed ends unconnected except by mesentery. Morley (1921) reviewed the literature concerning congenital occlusion of the ileum, and reported two examples.³

The patient was a male, age 5 days, admitted to hospital on March 21, 1930, with a history of vomiting since birth. The vomit was brown and feculent. One motion of small size was passed on March 19. The child lost weight, and was described as "ill, wasted, and dehydrated". On March 21 he vomited fecal matter twice during the night. No result followed the administration of two enemata, but a small amount of mucus was passed per rectum. Intestinal obstruction due to congenital malformation was diagnosed, but the child was too ill to submit to operation, and died on March 23.

Autopsy.—The body was that of a wasted male child. When the abdomen was opened the coils of small intestine were prominent and dilated, suggesting obstruction. The stomach and duodenum were not dilated, but the small intestine became increasingly distended as it was traced distally, ending in a bulbous extremity about two inches in diameter at a point nine inches from the ileocecal valve. The continuation of the ileum began as a

similar but much smaller blind end. Between them there was a wedge-shaped gap in the mesentery, the free border of which did not appear to contain any intestinal tissue. Distally, the ileum had but the diameter of a pencil, and passed upwards to join the cæcum in a normal manner. The drawing of the specimen (*Fig. 208*) illustrates these points clearly. Two enlarged lymphatic glands were present in the mesentery within the intestinal gap. The cæcum was displaced upwards and lay beneath the right lobe of the liver. The



FIG. 208.—Condition found at autopsy. A, Blind end of proximal ileum; B, Blind end of distal ileum; C, Appendix; D, Cæcum; E, Colon; F, Lymph gland; G, Gap in ileum.

appendix, cæcum, colon, and rectum were all narrow and atrophic. The colon, although less than a quarter of an inch in diameter, was perfectly formed. The anus was patent and admitted forceps to the rectum. Histological examination failed to demonstrate any intestinal tissue in the mesentery between the blind ends of the small intestine.

I wish to thank Professor C. W. Vining for permission to use his notes of this case, and Miss Collinson for the drawing.

REFERENCES.

- ¹ PASLEY, C. B., *Brit. Med. Jour.*, 1929, ii, 1056.
- ² FORSSNER, H., "Die angeborenen Darm- und Oesophagusatresien", *Anatomische Hefte*, 1907, xxxiv, 1, quoted by Morley.
- ³ MORLEY, J., *Brit. Jour. Surg.*, 1921-2, ix, 103.

UTERUS MASCULINUS :

COMPLETE TUBULAR HERMAPHRODITISM, WITH TERATOMATOUS ENLARGEMENT OF AN UNDESCENDED TESTICLE.

BY R. H. METCALFE,

SURGICAL REGISTRAR AT THE GENERAL HOSPITAL, BIRMINGHAM.

A YOUNG man (H. P. M.), 31 years of age, came to the out-patient department at the General Hospital, Birmingham, on April 2, 1930, complaining of abdominal pain and the presence of a large abdominal tumour arising out of the right iliac fossa.

EXAMINATION AND HISTORY.—The individual possessed all the external features of a well-developed man: he had a normal beard and growth of pubic hair; he had a bass voice; his penis was normal and well developed; there was no abnormal enlargement of the breasts. The only abnormal condition noted, apart from the presence of the abdominal tumour, was the fact that the testicles were undescended.

There had never been any doubt as to his sex—in fact, the individual had been married for seven years, and stated that he had had normal intercourse with his wife, with what he considered was a normal emission; the marriage, however, was childless, and the husband had gone so far as to have his wife examined by a gynaecologist.

In view of what was subsequently found, it is of interest to note that there was no history of menstruation or of monthly discomfort, neither was there any history of the passage of blood either per urethram or per rectum. There was an indirect hernia present on the right side, but this was easily reducible, and had never given rise to any trouble.

The patient was admitted to the General Hospital on April 7 under Mr. Seymour Barling, who performed a laparotomy on April 10.

OPERATION.—The abdomen was opened by a mid-line incision below the umbilicus: a large, smooth, bossy tumour presented itself and appeared to be free from adhesions, except for a few slight ones in the pelvis on the right side: these were freed, and the tumour was easily delivered outside the abdomen.

The tumour was found to be attached by a definite mesentery to the posterior surface of a structure which had the appearance of a broad ligament. Just above this was a tube with a fimbriated end, and these, on being traced towards the middle line, led to a rudimentary uterus, to the left side of which were attached a further Fallopian tube and broad ligament, normal in both size and position. An apparently normal 'ovary' was also found lying in its normal position on the left side. A complete excision of the upper part of the uterus and of both tubes and 'ovaries' was performed.

When the body of the uterus was divided, a canal about 1 cm. in diameter was found: a probe was passed down this canal for a distance of 15 cm. With a finger in the rectum, the tip of the probe was palpated

through the anterior wall of the rectum, lying in the mid-line just above a perfectly normal prostate. The probe itself could not be felt, and no communication between the uterine canal and the rectum was found. The raw areas in the pelvic floor were then peritonized and the abdomen was closed.

PATHOLOGICAL REPORT.—The excised specimen was examined in detail, and the general arrangement is well shown in the accompanying sketch (*Fig. 209*). The two tubular structures, seen lying in the angles between the uterus and the Fallopian tubes, had all the normal macroscopic appearances of vasa deferentia.

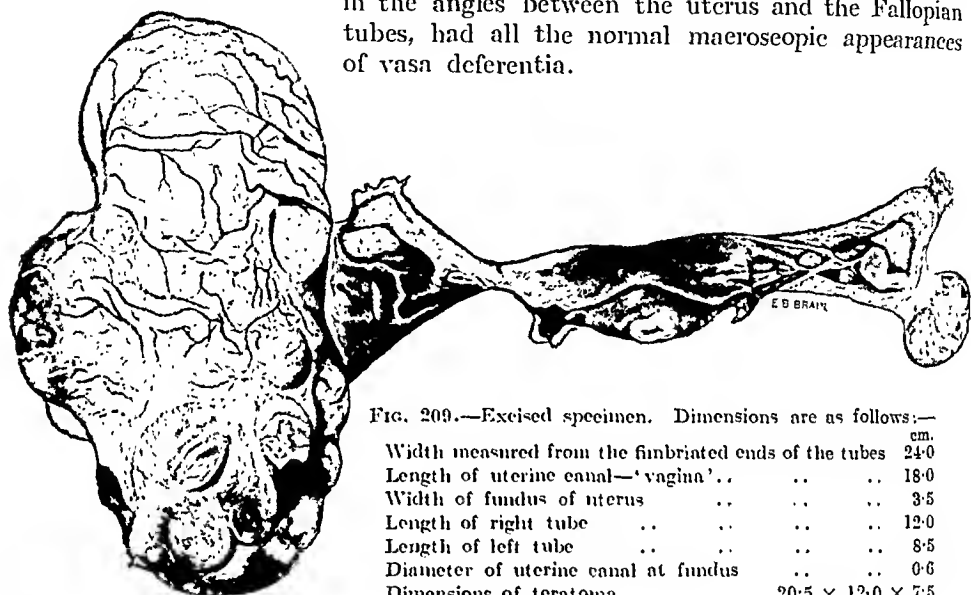


FIG. 209.—Excised specimen. Dimensions are as follows:—

	cm.
Width measured from the fimbriated ends of the tubes	24.0
Length of uterine canal—'vagina'..	18.0
Width of fundus of uterus ..	3.5
Length of right tube ..	12.0
Length of left tube ..	8.5
Diameter of uterine canal at fundus ..	0.6
Dimensions of teratoma ..	20.5 × 12.0 × 7.5
Dimensions of normal 'ovary' ..	3.0 × 2.4 × 1.6

As will be seen in the sketch, the whole arrangement of the uterus and tubes is exactly similar to that found in a normal female. The various parts of the specimen were cut and examined under a microscope. The following report was made:—

1. The tumour is composed of spheroidal cells and has a scirrhus form. The characters of the cells are those of an embryonal carcinoma of the testis.
2. The 'ovary' (?) is a testis, in which the interstitial cells are unduly prominent and the testicular tubules rather atrophic.
3. The tube near the angle of the uterus is a normal vas.
4. The lining of the uterine canal is tall columnar epithelium.

Cases of uterus masculinus have been recorded from time to time, but after a search of the literature, going back as far as 1855, no case has been found in which the reported female arrangement is as perfect as in the specimen described.

Langer¹ in 1855 reported a case in which the uterus and vagina measured 12.5 cm. in length—the 'ovaries', as in the present case, were mere testes. In 1888 the same author reported a second case, but the parts were smaller and

measured only 6 cm. in length. This case is of interest as normal vesiculæ seminales were found to be present; in practically all other cases these out-growths from the vas are not found.

Willett² reported a case in which the man was married, and his wife had children; but here, as in the last case, the parts were very rudimentary and measured only 4 cm. in length. Nearly all the other cases recorded have been sterile.

Primrose³ in 1898 reported a case very similar to the present one: here the patient complained of an abdominal tumour, which on laparotomy was found to be a teratoma of the testis lying attached to a rudimentary Fallopian tube. The uterus and vagina in Primrose's case measured 12 cm., as compared with 18 cm. in the present instance.

Several other cases of a similar nature are recorded, one being that of a French soldier in 1918,⁴ who was married and whose wife had borne children, and another being a child of 2 years of age, reported by Rutherford⁵ in 1929.

Cases of 'true hermaphroditism' occurring in man are found reported in the literature. In the majority of these, microscopical examination was not made of the glandular organs, and such cases must therefore be excluded: a few apparently conclusive cases remain.

It must be stressed that by 'true hermaphroditism' is meant a condition in which there exist in the same individual an ovary and a testis. This condition is what is known by the French authors as 'glandular hermaphroditism', as distinct from 'tubular hermaphroditism', the latter term being reserved to describe cases similar to the present one, and to those already quoted in this paper—that is to say, cases in which both the Müllerian and the Wolffian ducts persist in the same individual.

All degrees of 'tubular hermaphroditism' can occur, ranging from the rudimentary uterus masculinus, as normally found, to the complete persistence of the Müllerian ducts, as in the present case. This case is recorded as a perfect example of 'tubular hermaphroditism'.

I am indebted to Mr. Seymour Barling, Senior Surgeon to the General Hospital, Birmingham, for permission to publish details of this case.

REFERENCES.

- ¹ LANGER, "Ein neuer Fall von Uterus masculinus bei Erwachsenen", *Arch. f. Anat. u. Physiol.*, 1881.
- ² WILLETT, "Transverse Hermaphroditism in the Male", *Trans. Pathol. Soc. Lond.*, 1894, xiv.
- ³ PRIMROSE, "Uterus Masculinus in the Male", *Jour. Anat. and Physiol.*, 1898-9, N.S. xiii.
- ⁴ COUVELAIRE and DUCLUX, *Ann. de Gynécol. et d'Obst.*, 1918-19.
- ⁵ RUTHERFORD, "Case of Undescended Testicle with Uterus and Fallopian Tube", *Jour. of Anat.*, 1929, Oct., lxiv.

A RARE NECK CYST.

By IAN FRASER,

ASSISTANT SURGEON TO BELFAST HOSPITAL FOR SICK CHILDREN.

THE patient, Mrs. B., a widow aged 65, was referred to me for removal of a tumour in the neck.

HISTORY.—The swelling had appeared suddenly and painlessly five years previously and had remained constant ever since, and now she wished its removal for cosmetic reasons only.

ON EXAMINATION.—A smooth oval tumour, exactly the size of the average hen's egg, was felt. It was situated in the upper part of the posterior triangle of the neck. It lay deep to the deep fascia, and was freely movable in all directions with muscles either slack or tense. There were no other swellings or abnormalities present in the neck. The tumour was not affected by deglutition or posture, nor did it vary in size. It was absolutely painless, not tender, and the overlying skin was normal. Translucency could not be tested on account of its depth. On palpation the swelling was single, smooth, not lobulated, and if cystic was very tense, but it felt solid lying as it did partly under the trapezius. Sebaceous cyst, dermoid, branchial cyst, branchiogenic carcinoma, lipoma, and cystic hygroma were considered and excluded, and eventually it was agreed that the swelling must be a gland—perhaps tuberculous and perhaps undergoing calcification—though no focus in scalp, ear, or other drainage area could be found.

OPERATION (January, 1930).—An oblique incision three inches long was made over the swelling through skin, superficial fascia, and deep fascia. The tumour, which proved to be a tense cyst, shelled out with ease. It was thin-walled, and filled with white particles floating in a clear medium; the white bodies rapidly fell to the lower pole of the cyst. No diagnosis was made.

Fluid aspirated from the cyst showed it to be filled with active scolices, making the diagnosis of hydatid cyst definite. Further physical examination of the patient failed to reveal any suspicious masses in the liver or chest. An eosinophil count of 12 per cent confirmed the active nature of the parasite.

On subsequently questioning the patient I asked her had she ever been 'abroad'; after due consideration she said 'yes'—eight years ago she had been to Scotland for the day! A real item of interest in the case was the patient's extreme fondness for dogs. Her last pet (which with tears she explained had paid the supreme sacrifice at the hands of a reckless motorist) was now stuffed and retained as a pathological specimen in a glass case.

Comment.—Owing to the rarity of the condition in this country the case seems worth publishing. The points which it exemplifies are: (1) Hydatid cysts do occur sporadically in this country; (2) There is a close connection with the dog; (3) A single cyst can appear in the territory of the systemic

circulation without any signs in the portal area; (4) The cyst contents can remain highly active after five years.

I wish to record my thanks to Dr. Blake, who referred the case to me, and to Professor Drennan, Queen's University Pathological Department, who made an accurate diagnosis possible.

DERMOID CYST SIMULATING GASTRIC ULCER.

By R. J. McNEILL LOVE,

SURGEON TO ST. ANDREW'S HOSPITAL, POPLAR.

DERMOID cysts have been found in almost every part of the body, and when occurring in unusual sites are apt to lead to erroneous diagnosis.



FIG. 210.—Skiagram from a case of dermoid cyst simulating a gastric ulcer.
Note the opacity near the upper part of the greater curvature.

The following case is of interest in that the cyst in question simulated a gastric ulcer.

Mrs. E. C., age 53, was admitted to St. Andrew's Hospital, Poplar, after a brisk hæmatemesis, which was alleged to have exceeded a pint.

HISTORY.—The patient stated that for the past five years she had suffered from pain an hour to an hour and a half after food, but periodicity of symptoms

was indefinite. The pain was epigastric, and was relieved by the occasional vomiting with which it was sometimes accompanied. There was no loss of weight, and the general health was good. Two years previously she had been in hospital for a severe attack of pain accompanied with hæmatemesis.

ON EXAMINATION.—She was well nourished, and no physical signs could be elicited. Blood examination revealed a slight degree of secondary anaemia. A bismuth meal showed a normal stomach, but near the upper part of the greater curvature a curious opacity was noticed (*Fig. 210*). This was considered to be a calcified hydatid cyst, and although the patient had never been abroad, her residence in Poplar supported this diagnosis, as hydatid

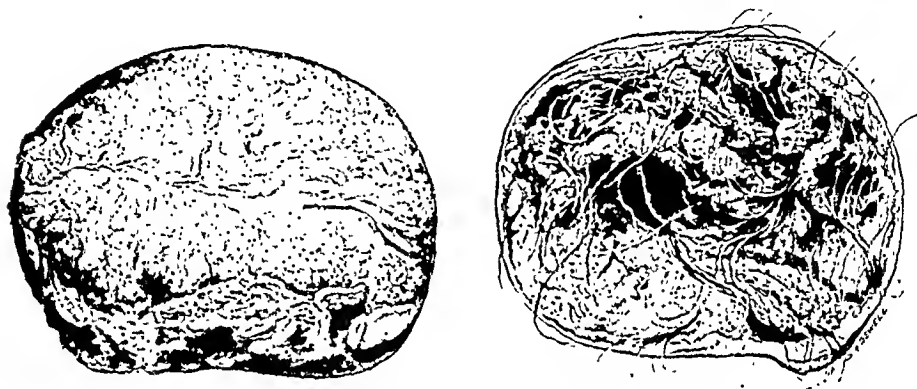


FIG. 211.—Dermoid cyst removed from the gastrosplenic omentum. The figure on the right shows the contents of the cyst. ($\times \frac{3}{4}$.)

cysts occasionally occur in this locality owing to the proximity of the docks. Calcification of the cyst was considered sufficient explanation for the absence of eosinophilia.

OPERATION.—As symptoms persisted a laparotomy was performed, and a hard rounded swelling about the size of a cricket-ball was discovered in the gastrosplenic omentum. This was easily shelled out, and proved to be a dermoid cyst, containing sebaceous material, rudimentary teeth, and hair (*Fig. 211*). All other viscera were healthy, and the hæmatemesis was presumably due to pressure on the vasa brevia veins.

Recovery was uneventful, and symptoms disappeared.

REVIEWS AND NOTICES OF BOOKS.

The Edwin Smith Surgical Papyrus. Published in Facsimile and Hieroglyphic Transliteration, with Translation and Commentary. By JAMES HENRY BREASTED. Demy 4to. Vol. I. Pp. 596 + xxiv, illustrated; Vol. II. 22 plates. 1930. Chicago: The University of Chicago Press. London: Cambridge University Press. 90s.

THESE sumptuous volumes should appeal to every surgeon, for the Edwin Smith Papyrus is the earliest known treatise on surgery. It is indeed incomplete at the beginning and at the end, but it gives details of forty-eight cases of injury to the head and neck, and is either the lecture notes of the surgeon who dictated it to the scribe, or the notes of his lectures taken by a pupil. At any rate, it seems to reproduce the very words of the author. The papyrus here copied was probably written in the sixteenth century B.C., and is itself a copy of a roll which may have been written a thousand years earlier, for many of the terms employed had already become obsolete, and the editor found it necessary to interpret them by a series of glosses. In like manner, an editor of to-day, copying a twelfth or thirteenth century English manuscript on anatomy or surgery, finds it necessary to explain many of the words used.

The original writer, who may have been contemporary with Imhotep, the priest-architect-physician when the Great Pyramid was being built, shows himself to have been a skilled surgeon who had seen war service. He had a scientific mind, was practical, and had few if any illusions as to the value of charms and amulets, although a later hand has attached to the copy of the roll a treatise on magic in the approved later style of Egyptian medicine.

The treatise shows that the writer followed a settled plan. He first gave a name to the injury; then made an examination, which it may be noted was always digital, and not by means of a probe or other instrument; offered a diagnosis; and afterward stated that the man could be cured, that an attempt could be made to cure him, or that nothing could be done. The treatment was either exclusively mechanical or surgical, as when a dislocated jaw was reduced in the same manner as it is to-day; a combination of purely surgical treatment with the use of external applications, as when splints were used made of wood and linen or of linen alone impregnated with glue and plaster; or by the employment of external applications only. It is interesting to observe that 'raw meat' was often applied as a dressing for wounds after they had been sutured. The use seemingly has never died out, for as a folk-remedy it was commonly used for a 'black eye', and in modern scientific surgery a piece of living muscle is used to arrest capillary hæmorrhage. It is recommended that the wounds should be stitched, closed by adhesive plaster—probably the diachylon or lead plaster still in use—or by bandages "obtained from the embalmers".

The roll shows some astounding physiological facts, unless we are reading our own knowledge into the words of this very ancient surgeon. After an injury to the head he directs that the pulse should be counted to know the condition of the heart, though he gives no indication that he recognized the circulation of the blood. He knew that there might be a depressed fracture of the skull without the skin's being broken, and that an injury to the brain was followed by loss of speech and paralysis of the limbs. He knew, too, that a dislocation of the vertebræ might lead to incontinence of urine and an emission of semen. There are indications that

aneurysms had occurred in his practice, for he speaks of soft swellings in the front wall of the chest and of similar swellings occurring in other parts of the limbs, adding wisely that their cure should not be undertaken. They might perhaps have been 'cold abscesses' due to tubercle, but these were familiar to Egyptian surgeons and would have been treated by them. Every case is a fascinating study from the surgical standpoint, but a just criticism should be used in their consideration.

Great praise must be given to the editor, Mr. Breasted, for his Foreword, general explanatory notes, and Introduction, all of which are full of information. The papyrus was bought at Luxor by Mr. Edwin Smith in 1862. Mr. Smith was probably the first American citizen to learn scientifically the little that was then known about the Egyptian language, and after his death the roll was presented by his daughter to the New York Historical Society. In 1922, being originally written in hieratic, it was transliterated into hieroglyphic characters, which is as much as to say that if it had been an early English manuscript it was transcribed into modern text hand. Of this transliteration an English version is given with a commentary in the first volume. The second volume reproduces in facsimile the original hieratic roll and the transliteration. The whole is appropriately dedicated to the memory of William Harvey on the three hundredth anniversary of the publication of *De Motu Sanguinis*, and forms the third and fourth volumes of the University of Chicago Oriental Institute Publications.

The printing has been done at the University Press, Oxford, and the plates have been produced by Emery Walker Ltd. Both firms are to be congratulated on the results.

Applied Anatomy. The Construction of the Human Body considered in Relation to its Functions, Diseases, and Injuries. By GWILYM G. DAVIS, M.D., Late Professor of Orthopaedic Surgery and Associate Professor of Applied Anatomy in the University of Pennsylvania. Revised by GEORGE P. MULLER, M.D., Professor of Clinical Surgery in the University of Pennsylvania. Eighth edition revised. Super Royal 8vo. Pp. 638 + xii, with 656 illustrations mostly from original dissections and many in colour by Edwin F. Faber. 1929. London: J. B. Lippincott Co. 42s. net.

A book of this magnitude, which has passed through eight editions since its first appearance in 1910, clearly requires no introduction or commendation. Its aim is not merely to relate anatomical facts, but to show the relations of structure to function and to describe the variations in structure which accompany diseased or injured conditions. It is in fact a text-book of surgery based chiefly on the anatomy of the body. There are obvious advantages and disadvantages in this plan of dealing with anatomy and surgery in the same book.

In the first place the practical essentials of anatomy are given without all the unnecessary minutiae which overload an ordinary text-book of anatomy; and then each surgical deformity or operation is described just when the anatomical picture of the part is fresh in the eye and mind. As against these advantages, however, there is the great drawback that surgery is considered only as a manipulative art, and, whilst the anatomical story runs consecutively, the surgical story is chopped up into little pieces. For example, in a book of this sort we might fairly look for an up-to-date account of the surgery of the sympathetic nervous system, but we look in vain. The sympathetic is not mentioned in the index, the cervical sympathetic and the description of its removal is found in the section on the neck, but we cannot discover any mention of the rami communicantes or of ramisection.

The present edition has appeared shortly after the death of Dr. Davis, whose great interest in orthopaedic surgery probably inspired the original idea of the work, because it is in this branch of surgery more than in any other that surgery and anatomy must go side by side.

The illustrations in number, originality, and excellence are such as to ensure the book a place in every reference library.

Die Chirurgie der Brustorgane. By FERDINAND SAUERBRUCH. Third edition. Vol. I. *Die Erkrankungen der Lungen.* Under the direction of H. ALEXANDER, H. CHAOUL, and W. FELIX. Part 2. *Die chirurgische Behandlung der Lungentuberkulose. Die parasitären Erkrankungen der Lungen. Lungentumoren. Die chirurgische Behandlung des Asthma bronchiale. Syphilis der Lungen.* Imperial 8vo. Pp. 1373 + viii, with 189 illustrations. 1930. Berlin: Julius Springer. RM. 98.

The first part of this monumental work has already been reviewed in this JOURNAL. The second part deals chiefly with the surgical treatment of pulmonary tuberculosis, and forms a valuable monograph on this subject. The distinguished author is assisted by Drs. Alexander, Chaoul, and Felix.

The opening chapters deal with the history of the subject, the usual course of pulmonary tuberculosis, and the changes in the lung and chest which occur when natural recovery takes place. The natural falling in of the chest wall and the shrinking of the lung form the basic ideas upon which surgical treatment is founded.

The subject of artificial pneumothorax is then described and discussed in some detail. The theoretical aspect is shortly dealt with and is well illustrated by X-ray pictures, which chiefly show the outline of the contracted lung on the side of the pneumothorax. The pathological anatomy of the method is very convincingly described by figures of the collapsed lung and eleven microscopical preparations, beautifully reproduced in colour, showing the healing process taking place in such an organ. The technique of the operation by puncture or by incision is given, and the method of dividing adhesions under the guidance of thoracoscopy (Jacobeus). The author, however, considers that this complication in technique makes the liability to exudate or emphysema greater, and therefore condemns it. Among the more important complications after pneumothorax are mentioned and illustrated interstitial emphysema, subdiaphragmatic air collection, air embolism, and pleurisy.

In summarizing the results of this method figures are quoted which show that when there are no adhesions 70 per cent of the patients are cured (periods of three to thirteen years). Patients with localized adhesions give only 33 per cent of recoveries, whilst those with wide adhesions show only 11 per cent. The various operations for diminishing the capacity of the chest wall are described and very clearly illustrated. The most complete of such operations (Bauer-Friedrich), in which the greater part of the ribs from the 2nd to the 10th or 11th are removed, is rightly relegated to small print, and the chief stress is laid upon the paravertebral resection of a long piece of all the ribs from the first to the 11th. This is done in two stages, the upper ribs being left for the second operation. There are a number of good photographs and X rays of patients. After this thoracoplasty other operations—e.g., the insertion of fat grafts or paraffin between the collapsed lung and the chest wall, and avulsion of the phrenic nerve—are well described, but the author evidently does not regard such procedures with much favour. One of the most valuable chapters in the book is that in which the indications for surgical treatment of pulmonary tuberculosis are discussed and the importance of co-operation between surgeons and physicians is stressed. There are short chapters dealing with a classified bibliography which occupies 169 pages.

This work, which forms a standard book of reference, reflects great credit on the author and his assistants and on the publisher.

A Text-book of Orthopedic Surgery. By WILLIS C. CAMPBELL, M.D., F.A.C.S., Professor of Orthopedic Surgery, University of Tennessee College of Medicine, etc. Medium 8vo. Pp. 705, with 504 illustrations. 1930. Philadelphia and London: W. B. Saunders Co. 37s. 6d. net.

It is somewhat difficult to assess the real object of the author in writing this book. It attempts to cover a very wide range, and consequently varies greatly in the amount of detail in the information provided: on the one hand, many orthopaedic conditions

receive a shorter and less complete description than they do in the average text-book of general surgery; on the other hand, certain methods of treatment are dealt with at a length that is of value only to the orthopædic specialist—yet such conditions as congenital club-foot and congenital dislocation of the hip receive only scant attention.

The illustrations are numerous, but a number of them appear to serve little purpose—for instance, in the section on fractures there are many X rays taken before and after treatment, but scarcely any illustrations of methods of treatment. The descriptions of methods of fracture treatment are, themselves, very uneven. Incidentally, a pin $\frac{1}{4}$ in. in diameter is advised for skeletal traction.

The classification of certain conditions of the joints together as low-grade affections seems to serve no useful purpose. They include infectious arthritis, progressive polyarticular and hypertrophic arthritis, gout, syphilis, trophic arthropathies, osteochondritis, endocrine gland disturbances, hæmophilia, neoplasms, and tuberculous arthritis. Similar conditions of the bones appear under the classification of constitutional affections (with the exception of neoplasms), although they are called low-grade affections in the first chapter. This somewhat curious classification goes with a general lack of pathological description that is in evidence throughout the book. It is unlikely that this work will appeal to any group of English readers.

Research and Medical Progress and other Addresses. By J. SHELTON HORSLEY, M.D., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va. Crown 8vo. Pp. 208. Illustrated. 1929. London: Henry Kimpton. 8s. 6d. net.

THIS is a collection of addresses, mostly of a presidential character, delivered to medical societies and previously published in the medical journals: they are now assembled in book form, the volume being dedicated to the ex-interns of St. Elizabeth's Hospital, Richmond, Virginia. They deal, in the main, with the applications of modern research in physiology and other ancillary sciences to medicine and surgery, with the career and ideals of the modern surgeon, and with the relations of the medical profession to politics and to public authorities. Collectively they constitute a volume of extremely interesting reading, in which the author's opinions on many diverse subjects are concisely and forcibly expressed, and are such as will meet with wide approval. We in this country will appreciate the prominence given to the career of Sir James Mackenzie as illustrative of successful research. On page 41 it is stated that Sir James solved many of his earlier problems "in the isolated Scotch village of Burnley"—an error which must make a quite unintentionally humorous appeal to the 104,000 inhabitants of that typical Lancashire town.

La Rachianesthésie: sa Valeur et sa Place actuelle dans la Pratique. By EMILE FORGUE, Professeur de Clinique chirurgicale à la Faculté de Montpellier; and ANTOINE BASSET, Professeur agrégé à la Faculté de Médecine de Paris: Chirurgien des Hôpitaux. Medium 8vo. Pp. 222, with 23 illustrations. 1930. Paris: Masson et Cie. Fr. 30.

ALTHOUGH both authors have collaborated to produce this monograph, yet since each has written his allotted sections quite independently, some overlapping is only to be expected: they are in mutual agreement, however, in their conclusions. M. Forgue is responsible for the physiology, indications and contra-indications, advantages, and technique of rachianæsthesia, while M. Basset has written of the failures, accidents during and after its administration, their prophylaxis and treatment, and their pathogeny and mortality. Great stress is laid upon the temporary intoxication of the *entire* organism that necessarily accompanies general anæsthesia, more especially its effects upon the functions of the liver and kidneys; and the advisability of minimizing these undesirable effects constitutes a strong argument in favour of local or spinal anæsthesia. A full account is given of the history of spinal anæsthesia,

starting from its accidental discovery by Corning, of New York, in 1885. Its physiology, indications, and technique are thoroughly and judiciously discussed, and the operative details are well illustrated by diagrams and photographs. Preference is given to novocain over the other spinal anæsthetic drugs, but 'percein', so much in evidence just now, is of too recent introduction for discussion in this monograph, nor is anything more than a passing reference made to Pitkin's method of controllable spinal anæsthesia. The uncommon but extremely interesting complication of paralysis of the sixth cranial nerve following upon spinal anæsthesia is described at length, and it is pointed out that eventual recovery is the rule, though one case lasted as long as eight months. An excellent bibliography is appended.

Die Knochenbruchbehandlung mit Drahtzügen. By Prof. Dr. RUDOLF KLAPP, Direktor der chirurgischen Universitätsklinik, Marburg a.d. Lahn.; and Dr. WERNER BLOCK, Chefarzt der chirurgisch-gynäkologischen Abteilung am Marienhospital, Witten-Ruhr. 10" x 7". Pp. 294. Illustrated. 1930. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 25; bound. RM. 28.

It is remarkable that a monograph of this size should have been devoted to so small a subject as the treatment of fractures by means of traction on a transfixion wire. Almost half the book is concerned with a description of different varieties of the wire traction apparatus and its method of application. Perhaps one might have been glad to be given a little less in this section, because so many different forms of apparatus are described that the essential simplicity of the method is lost sight of. In its latest and most refined type the method is as follows. The wire is hardened steel 0.75 mm. thick such as is used for pianos. It is inserted by means of Kirschner's apparatus. A given length of wire has one end sharpened and the other is held by a revolving motor. The piece of wire about six inches long is held rigid in a frame which collapses as the wire is driven into place. By this means no separate drill is required. The wire having transfixed the limb, its two ends are held in a special horseshoe-shaped clamp in which it is fixed under light tension. It is claimed that there is so little damage to the tissues that septic infection or sinus can never occur. The great *a priori* objection to this method is that a fine wire to which a traction weight of 25 lb. is attached would tend to travel through the bone. This difficulty is met by saying that in actual practice such a misadventure does not occur, and it is supposed that a reactive inflammation takes place in the bone round the wire track which prevents the wire from pulling through. Experimental specimens are shown, however, in which the wire has actually travelled for some distance. Another definite objection to the wire as compared with the transfixion pin is that the apparatus for its insertion and traction is definitely more complicated.

The remainder of the book is occupied with a systematic description of the application of the wire traction to various fractures. This part might equally well have been written about any efficient method of skeletal traction. We think that the more complicated forms of apparatus in which double transfixion and lateral pulls are combined are not to be recommended for ordinary fracture treatment; but the clear exposition of this newest method of skeletal traction is very valuable for purposes of reference.

The Dramatic in Surgery. By GORDON GORDON-TAYLOR, O.B.E., M.A., F.R.C.S., Surgeon to the Middlesex Hospital. Medium 8vo. Pp. 88. Illustrated. 1930. Bristol: John Wright & Sons Ltd. 12s. 6d. net.

This little book represents the substance of an address given by the author to the Manchester Surgical Society. We venture to think that the address has lost and not gained by being committed to cold print, because disconnected accounts of

wonderful cases need the living personality of the narrator to make a dramatic whole. On the other hand, we welcome the book as giving some beautiful coloured illustrations of remarkable specimens which otherwise might have remained unpublished—such are the chondroma of the ilium, the myeloma of the innominate, and the resected half jaw with an epithelioma. The book, although abounding with personal experiences, is by no means confined to them, and full credit is given to various surgeons who have dared and done great surgical feats.

Radium in General Practice. By A. JAMES LARKIN, B.Sc., M.D., D.N.B., Radium Consultant on Staffs of Wesley Memorial, German Evangelical Deaconess, John B. Murphy, Washington Park Community Hospitals, Chicago, etc. Medium 8vo. Pp. 304 + xiii, with 28 illustrations. 1929. New York: Paul B. Hoeber Inc. \$6.00.

THE chief object of this book is to make the general practitioner of medicine conversant with the scope of and indications for the treatment of various general and local diseases by radium. The theoretical and technical aspects of the subject are presented in the barest outline, whereas the clinical aspects are dealt with at some length. It is a very well balanced account of the subject, without making any extravagant claims. The general dangers of and contra-indications to the use of radium are clearly stated at the outset, and special points for caution are repeated under each disease. The principal general diseases discussed are Banti's disease, Hodgkin's disease, hyperthyroidism, and leukaemia. In each of these it is considered that radium is a powerful remedial agent. The other sections are related to various gynaecological conditions, chiefly uterine fibroids and malignant disease; various tumours, simple and malignant; and miscellaneous skin diseases. Short case histories are given illustrative of each condition. These might well have been fuller and more detailed. A considerable list of articles on the subject is added to each chapter. We consider that the author has given a clear, fair, and critical account of the modern use of radium as a therapeutic agent, and that it will be of great value to student, practitioner, and specialist, both for reference and as an introduction to the subject.

L'Ostéo-synthèse métallique dans les Fractures diaphysaires. Etude expérimentale et anatomo-pathologique. Applications pratiques à la Chirurgie humaine By ROBERT FRANTZ, Ancien Interne des Hôpitaux de Paris. With a Preface by Professeur BERNARD CUNÉO. Royal 8vo. Pp. 190. Illustrated. 1929. Paris: Masson et Cie. Fr. 35.

THIS is a record of experiments on dogs to ascertain the effect of various metallic contrivances used in the operative treatment of fractures. They are concerned mainly with the form of the apparatus rather than with the nature of the metal. Eleven dogs were operated upon; in four the bone was not fractured, but a screw, a Lambotte plate and screws, Cunéo's staple and wire, and a Hallopeau plate fixed by Parham's bands were used respectively. The histological results are described in considerable detail and show that the compact bone in the region of the metal undergoes rarefaction, and that if the medulla is reached a marked fibrous reaction occurs round the metal.

In the other seven dogs the bone was broken and then fixed in various ways. Three of these cases did not heal satisfactorily, so that only four dogs with six fractures were available for study, there being two fractures treated with Cunéo's staple and wire, Hallopeau's plate with Parham's bands, and Contremoulin's perforated aluminium-bronze sleeve respectively.

The deductions drawn are that the fragments should be securely fixed and that the apparatus should spread its force over a considerable area of bone so that the pressure should not be too great at any one point, and that for this purpose the Contremoulin sleeve is best adapted.

The first point has already been decided by much experience in the treatment of fractures in man, and the evidence in these experiments is insufficient to show that any one of the methods here used is the best. With regard to the second point, the Contremoulin sleeve undoubtedly secures a wide area of application, and does not, unless too tightly applied, damage the underlying bone and medulla, but it does limit the formation of the external callus opposite the fracture.

L'Arthrite chronique de la Hanche. By L. DUVERNAY, Médecin consultant à Aix-les-Bains. Medium 8vo. Pp. 145, with 51 illustrations. 1930. Paris: Masson et Cie. Fr. 40.

This monograph is a concise and readable account of chronic arthritis of the hip-joint based on the considerable experience of the writer as a physician at Aix. Although it contains little original matter, there is much that will interest the orthopaedic surgeon. The writer divides chronic arthritis of the hip into two main groups: (1) Where arthritis develops in a hip previously normal; (2) Where the arthritis is superimposed in a hip-joint deformed since childhood as a result of congenital subluxation, coxa plana, or coxa vara. In the clinical picture the author stresses four early signs: (1) Limitation of abduction and adduction; (2) The 'shoe' sign—that is, inability of the patient to take off his shoe in the ordinary way; (3) Pain referred to the inguinal region; and (4) Relatively free range of flexion until the later stages. The surgical treatment is briefly discussed. The author's preference is for arthrodesis, if the patient is fit to undergo the fairly severe ordeal which this operation entails. The advantages of reconstructive excision of the femoral head do not appear to be familiar to the author.

Die Wirbelbrüche und ihre Endergebnisse. By Dr. WALTER HAUMANN, Medical Superintendent of the Evangelical Hospital, Hattingen in Ruhr. Super royal 8vo. Pp. 180, with 20 illustrations. 1930. Stuttgart: Ferdinand Enke. Paper covers, RM. 19; bound, RM. 21.

This monograph is one of considerable value because it gives a critical and analytical account of over two hundred consecutive cases of fracture of the vertebrae. The distribution of the lesions was: cervical 10, dorsal 56, lumbar 138. The spinal cord was affected in 100 per cent of the cervical fractures and in only 50 per cent of those of the dorsal and lumbar regions; 71 per cent were caused by indirect violence, and 29 per cent by direct. The vertebra most often injured was the 1st lumbar, then the 2nd lumbar, and then the 12th dorsal. Certain generalizations are made. Cord lesions are held to be a contra-indication for operation, and the value of a stabilizing bone-graft or ankylosing operation for cases without nerve lesion is hardly recognized. No case of late development of symptoms (Kümmel's disease) was observed, neither was there a single case in which tuberculous disease followed injury. Local spondylitis deformans, however, was comparatively common. Special attention is given to cases of exceptional interest—for example, one case of unilateral cord lesion with Brown-Séquard syndrome, and one of double brachial plexus lesion, both these being in fractures of the cervical region.

Diabetic Surgery. By LELAND S. McMITTRICK, M.D., F.A.C.S., Visiting Surgeon, Palmer Memorial Hospital; and HOWARD F. ROOT, M.D., Assistant Physician, New England Deaconess Hospital. 9½" x 6". Pp. 269 + ix, with 79 illustrations in the text and 2 plates. 1929. London: Baillière, Tindall & Cox. 21s. net.

THE worst thing about this book is its title, and we trust that the authors will see their way to altering it to "Surgery of Diabetes" in the next edition—for the book is of great value and we feel sure that another edition will soon be needed. The

main object of the work is to correlate the treatment of diabetes by diet and insulin with that of surgical operation. Before the discovery of insulin it was rightly held that many cases of diabetes requiring surgical treatment presented so grave a risk that coma and death would probably supervene. Now insulin therapy has changed all this, and there is hardly ever a case in which the advantages of surgery need be denied to the diabetic. The tabulation of suitable diets and the dosage of insulin in relation to such diet is, we think, the most valuable feature of the book.

The largest section is devoted to a discussion of gangrene and the allied lesions of the lower extremities. This includes a careful discussion of the distinction between arteriosclerotic and septic types of gangrene and the determination of the proper site of amputation in each case. We think that some reference ought to be made to the nerve lesions, e.g., neuritis, and to arterial spasm, with the allied question of the rôle of sympathectomy.

The other chapters deal with gangrene of the upper extremities including whitlows, carbuncles, abdominal surgery, thyroid diseases, and malignant disease. The book concludes with a useful list of technical methods.

Die Chirurgie, A System of Surgery. Edited by Profs. M. KIRSCHNER (Tübingen) and O. NORDMANN (Berlin). Royal 8vo. Berlin and Vienna: Urban & Schwarzenberg.

FASCICULUS 26 (Vol. II). Pp. 1583-1828, with 175 illustrations in the text and one coloured chart. 1930. RM. 18.

THIS section, which deals with the surgery of the bones, is by Dr. Leopold Schonbauer, of Vienna. In regard to the completeness of its subject-matter, the wealth of original illustrations, and the extensive references to literature, it is excellent, and forms a most valuable work of reference.

The section begins with a short but clear summary of our knowledge of the anatomy and physiology of bones, discussing the various conditions which influence growth and development. Then follows a short account with good illustrative figures of the chief congenital malformations, especially the so-called congenital amputation. Various theories about the rôle of the periosteum and the causal agents in bone regeneration are stated, but we are surprised that the work of Macewen is not referred to. The section on osteomyelitis is rather disappointing owing to its brevity. The vexed question as to whether incision of the periosteum is sufficient treatment of the acute disease or whether the medulla should always be opened is not definitely answered, but the balance of the arguments given is in favour of the former procedure.

The sections on osteitis fibrosa, osteitis deformans, and the new growth of bone are notable for the wealth of good illustrations. From a practical point of view it would perhaps have been of greater value if more of the early stages and less of the late stages of these diseases had been described. The section on fractures and their treatment is too brief to be of much practical value. Operative treatment and bone-grafting are hardly mentioned.

The chief value of this book to English readers is found in the good illustrations of rare and unusual diseases and the extensive lists of references to Continental authorities.

FASCICULUS 27 (Vol. III). Pp. 609-832, with 171 illustrations. 1930. RM. 14.

THE present section by Dr. E. Heymann, of Berlin, is confined to a consideration of the diseases of the spinal cord and its membranes. The introductory chapter contains a generous appreciation of the part taken in the foundation of the surgery of the spinal cord by Macewen and Horsley. The account of the anatomy and physiology of the cord is very clear and complete, and is enriched by many valuable diagrams showing the segmental distribution of the nerves. One of these of the

innervation of the viscera would make the monograph worth having, even if it contained nothing else. There are also full and well illustrated accounts of the tumours of the cord, of the operation of chordotomy, and the various ways of exposing the spinal cord. This and the section on the peripheral nerves are among the best monographs in this work.

FASCICULUS 28 (Vol. III). Pp. 833-980, with 71 illustrations in the text, many in colour, and 2 coloured plates. 1930. RM. 11.

THIS section by Professor Lebemann (Frankfort-on-Main) is devoted entirely to the surgery of the peripheral nerves. It begins with general considerations relating to the anatomy of normal nerves and the results of their division. Interesting statistics are given of the frequency of injury to individual nerves. Thus the first four nerves in order of frequency in regard to injury are the ulnar (158), the musculospiral (129), the median (117), and the sciatic (55). The importance of early operation is shown by a table in which the percentage of successes after suture was 100 in cases done within two months of injury, 62 within six months, and 25 within twelve months. It is considered that suture is worth while up to two years after the division.

In describing various operative procedures for making good defects in the nerve it is probable that undue prominence is given to ingenious devices which have little or no practical value, but these methods are well described and illustrated.

The section devoted to the special nerves is good and helpful—for example, the figures of the exposure of the sensory root of the trigeminal nerve are very clear, and the muscle substitution operations for facial paralysis are equally well given, as are those for dealing with a cervical rib. The subject of the peripheral nerves does lend itself to treatment in a small and compact monograph such as this, and we think that the author has done justice to the subject. Also he has been much more catholic in his appreciation of workers outside Germany than many other contributors to this great system of surgery which is now nearing completion.

Handbook on Tuberculosis. By B. S. KANGA, M.D., D.P.H., Medical Officer, Turner Dispensary, and Visiting Medical Officer, Turner Sanatorium (Bombay Municipality). Crown 8vo. Pp. 150 + viii. Illustrated. 1930. London: John Bale Sons & Danielsson. 5s. net.

THIS handbook is a short and clearly written account of tuberculosis of such an elementary character as to make it suitable for propaganda purposes amongst non-medical readers. It contains a strong plea for proper organization of prophylactic measures for the prevention of the disease and of a system of sanatoria and dispensaries for its treatment. Questions of surgical treatment are only mentioned very briefly.

Die Wiederbelebung: Eine zusammenfassende Darstellung ihrer Theorie und Praxis. By Prof. Dr. OSKAR BURNS and Dr. KARL THIEL, der Medizinischen Universitäts-Poliklinik, Königsberg. Super royal 8vo. Pp. 109, with 26 illustrations. 1930. Berlin and Vienna: Urban & Schwarzenberg. RM. 9.

THE authors consider that, owing to the rapid advance of industrial machinery and electricity as well as to the increased number of motor accidents, there is a marked increase in the number of cases of loss of consciousness with temporary cessation of respiration and circulation in which restorative methods may be necessary. Within the compass of a very short and practical monograph is given a complete account of the whole subject of the signs of death and the methods of restoration of life, together with a sufficient reference to theoretical considerations and scientific experiment to enable the reader to get a practical knowledge of the subject or to pursue any special branch of it in further detail.

After a general section concerned with the restoration of circulation and respiration, a special section follows dealing with special types of death, e.g., that by freezing, by heat stroke, by electrical currents, poisoning by carbon monoxide, benzine, alcohol, morphia, and various anæsthetics and narcotics. The book concludes with a list of references to literature dealing with these subjects.

The Clinical Pathology of Thoracic Puncture Fluids. By S. Roodhouse GLOYNE, M.D., D.P.H., Pathologist, City of London Hospital for Diseases of the Heart and Lungs, Victoria Park. Post 8vo. Pp. 83 + vii. Illustrated. 1930. London: John Bale Sons & Danielsson. 10s. 6d. net.

This small book is a practical attempt to aid diagnosis in early and obscure diseases of the chest by means of examination of the fluids withdrawn from the pleural cavity by an exploring syringe. Chief reliance is placed upon the number and character of the cells, but bacteriological examination and fixation tests also are used. It is considered that the great majority of acute serous pleural effusions are of tuberculous nature. In the case of empyema, the nature and number of the organisms found is a valuable guide not only to treatment but also to prognosis.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Shorter Convalescence. By Lieut.-Colonel JAMES K. McCONNEL, D.S.O., M.C., Member of the Chartered Society of Massage and Medical Gymnastics; Bio-physical Assistant (working at St. Thomas's Hospital). With a Foreword by Sir ROBERT JONES, Bart., K.B.E., C.B., F.R.C.S. Crown 8vo. Pp. 132 + xi. Illustrated. 1930. London: William Heinemann (Medical Books) Ltd. 5s. net.

Collected Papers of the Mayo Clinic and the Mayo Foundation. Edited by Mrs. M. H. MELLISH, RICHARD M. HEWITT, M.A., M.D., and MILDRED A. FELKER, B.S., Vol. XXI. 1929. Medium 8vo. Pp. 1197 + xxiii, with 276 illustrations. 1930. Philadelphia and London: W. B. Saunders Co. 60s. net.

Surgical Diagnosis. By American authors. Edited by EWARTS AMBROSE GRAHAM, A.B., M.D., Bixby Professor of Surgery, School of Medicine, Washington University, St. Louis, etc. Volume III. 9½" × 6". Pp. 1043, with 446 illustrations. 1930. Philadelphia and London: W. B. Saunders Co. Per set—three volumes and index volume, £7 10s.

Physiologie, pathologique chirurgicale. Inflammations, Effects des Traumatismes, Reparation des Plaies, Greffes, Maladies des Os, des Articulations, des Vaisseaux et des Nerfs. By R. LERICHE, Professeur de Clinique chirurgicale à la Faculté de Strasbourg; and A. SOLICARD, Professeur d'Histologie à la Faculté de Lyon. Medium 8vo. Pp. 212. 1930. Paris: Masson et Cie. Fr. 26.

Plarr's Lives of the Fellows of the Royal College of Surgeons of England. Revised by Sir D'ARCY POWER, K.B.E. (Mil.), F.R.C.S., Hon. Librarian Royal College of Surgeons; with the assistance of W. G. SPENCER, O.B.E., M.S., F.R.C.S., and Professor G. E. GASK, C.M.G., D.S.O., F.R.C.S. In two volumes. Royal 8vo. Vol. I: Pp. 752 + xxvi. Vol. II: Pp. 596. 1930: Printed and published for the Royal College of Surgeons by John Wright & Sons Ltd., Bristol. Cloth, 42s. net; half bound, 57s. 6d. net.

REVIEWS AND NOTICES OF BOOKS 351

- La Sacro-Coxalgie et son Traitement.** By PIERRE INGELBANS, Chef de Clinique de Chirurgie Infantile à la Faculté de Médecine de Lille. Preface by RENÉ LE FORT. $9\frac{1}{2}'' \times 6\frac{1}{4}''$. Pp. 140, with 6 plates. 1930. Paris: Masson et Cie. Fr. 30.
- La Pratique chirurgicale illustrée.** By VICTOR PAUCHET. Fasc. XVI. Super royal 8vo. Pp. 220, with 175 illustrations drawn from nature by S. Dupret. 1930. Paris: G. Doin et Cie. Fr. 65.
- On Faith and Science in Surgery.** By Sir JOHN BLAND-SUTTON, Bart., Vice-president and Consulting Surgeon to the Middlesex Hospital. Post 8vo. Pp. 109 + xii. Illustrated. 1930. London: William Heinemann (Medical Books) Ltd. 7s. 6d. net.
- Gonococcal Infection in the Male.** By ABR. L. WOLBARST, M.D., Urologist and Director of Urologic Clinics, Beth Israel Hospital, etc. Second edition, completely revised and enlarged. Medium 8vo. Pp. 297, with 140 illustrations, including 7 colour plates. 1930. London: Henry Kimpton. 25s. net.
- Ueber die schmerzhafteste Schulterversteifung (Periarthritis humeroscapularis).** By Prof. Dr. E. SEIFERT, Oberarzt der chirurgischen Univ.-Klinik, Würzburg. $9\frac{1}{2}'' \times 6\frac{1}{4}''$. Pp. 283-317, with 13 illustrations. 1930. Leipzig: Curt Kabitsch. RM. 4.
- Guy's Hospital Reports.** Edited by ARTHUR F. HURST, M.D. Vol. LXXX (Vol. X Fourth Series), No. 3. July, 1930. Royal 8vo. Pp. 253-378. Illustrated. 1930. London: The Lancet Ltd. Single numbers, 12s. 6d. net; annual subscription, £2 2s. net.
- Die spezielle Chirurgie der Gehirnkrankheiten.** Three volumes. Vol. I, compiled by Dr. K. BOSHAMER, Prof. GUSTAV BRÜHL, Prof. N. GULEKE, Dr. E. HARMS, Dr. G. JORNS, Prof. HANS SMIDT, Prof. C. TH. WILICH. Edited by Prof. FEDOR KRAUSE. Royal 8vo. Pp. 735 + xxiv, with 41 illustrations. 1930. Stuttgart: Ferdinand Enke. Paper covers, RM. 96; bound, RM. 99.
- The Metabolism of Tumours.** Investigations from the Kaiser-Wilhelm Institute for Biology, Berlin-Dahlem. Edited by OTTO WARBURG, Kaiser-Wilhelm Institute for Biology, Berlin-Dahlem. Translated from the German edition with accounts of additional recent researches, by FRANK DICKENS, M.A., Ph.D., whole-time worker for the Medical Research Council, Courtauld Institute of Biochemistry, Middlesex Hospital, London. Demy 8vo. Pp. 327 + xxiv. Illustrated. 1930. London: Constable & Co. Ltd. 40s. net.
- Lehrbuch der Mund- und Kieferchirurgie.** By Prof. Dr. ERICH SONNTAG, A.O. Planmäßiger Prof. der Chirurgie und Direktor des Chirurgisch-Poliklinisch Instituts der Universität Leipzig; and Prof. Dr. WOLFGANG ROSENTHAL, A.O. Prof. der Chirurgie an der Universität Leipzig. Royal 8vo. Pp. 444 + xv, with 501 illustrations. 1930. Leipzig: Georg Thieme. Paper covers, M. 24; bound, M. 26.
- Surgical Pathology and Morbid Anatomy (Bowlby and Andrews).** Revised by GEORGE KEYNES, M.A., M.D., F.R.C.S., Assistant Surgeon, St. Bartholomew's Hospital. Eighth edition. Medium 8vo. Pp. 644 + x, with 224 illustrations. 1930. London: J. & A. Churchill. 21s. net.
- Minor Surgery and Bandaging For the Use of House Surgeons, Dressers and Junior Practitioners.** By GWYNNE WILLIAMS, M.S., F.R.C.S., Surgeon, University College Hospital. Twentieth edition. Crown 8vo. Pp. 445 + viii, with 2621 illustrations. 1930. London: J. & A. Churchill. 10s. 6d. net.
- The Science and Practice of Surgery.** By W. H. C. ROMANIS, M.A., M.B., M.Ch. Cantab., F.R.C.S., F.R.S. (Edin.), Senior Surgeon in Charge of Out-patients, Surgeon to the Isolation Block, and Teacher of Practical Surgery, St. Thomas's Hospital, etc., and PHILIP H. MITCHELL, M.D., M.S. (Lond.), F.R.C.S., Surgeon in charge of Out-patients, Teacher of Operative Surgery, and Demonstrator of Anatomy, St. Thomas's Hospital, etc. Third edition. In two volumes, with 716 illustrations. Vol. I, General Surgery. Pp. 772 + x. Vol. II, Regional Surgery. Pp. 950 + x. 1930. London: J. & A. Churchill. 14s. each volume.

Manual of Surgery for Students and Practitioners (Rose and Carless). By CECIL P. G. WAKELEY, F.R.C.S., F.R.S. Edin., Erasmus Wilson Lecturer, Royal College of Surgeons of England; Surgeon, King's College Hospital, etc.; and John B. HUNTER, M.C., M.Chir., Cantab., F.R.C.S., Assistant Surgeon, King's College Hospital, etc., Thirteenth edition. $9\frac{1}{2}'' \times 6\frac{1}{2}''$. Pp. 1592 + xii, with 664 illustrations in the text, 19 coloured, and X-ray supplement. 1930. London: Baillière, Tindall & Cox. 30s. net.

Die Chirurgie. Edited by Profs. M. KIRSCHNER (Tübingen), and O. NORDMANN (Berlin). Fasc 29 (Vol. II). Royal 8vo. Pp. 1829-2028, with 98 illustrations. 1930. Berlin and Vienna: Urban & Schwarzenberg. RM. 16.

Die Mund- und Halsoperationen. By Prof. Dr. J. SOERRESEN (Berlin). Super royal 8vo. Pp. 457 + x, with 42 black-and-white illustrations and 48 coloured plates. 1930. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 85; bound, RM. 95.

The Student's Handbook of Surgical Operations. By Sir FREDERICK TREVES, Bart., G.C.V.O., C.B., LL.D., F.R.C.S., revised by CECIL P. G. WAKELEY, F.R.C.S.Eng., F.R.S. Edin., Surgeon to King's College Hospital, etc. Fifth edition. Crown 8vo. Pp. 535 + xi, with 190 illustrations. 1930. London: Cassell & Co. Ltd. 10s. 6d. net.

Cancer of the Lung and other Intrathoracic Tumours. By MAURICE DAVIDSON, M.A., M.D., B.Ch. (Oxon.), F.R.C.P. (Lond.), Physician to the Brompton Hospital for Diseases of the Chest, etc. With a Foreword by ARTHUR J. HALL, M.A., M.D., D.Sc., F.R.C.P. (Lond.), Professor of Medicine, University of Sheffield. Super royal 8vo. Pp. 173 + x, with 62 illustrations. 1930. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

THE BRITISH JOURNAL OF SURGERY

VOL. XVIII.

JANUARY, 1931.

No. 71.

SOME BYGONE OPERATIONS IN SURGERY.

BY SIR D'ARCY POWER, K.B.E., LONDON.

III. CUTTING FOR THE STONE *(continued)*.

CHESELDEN'S OPERATION.

JOHANNES JACOBUS RAU (1668-1719) was born in Baden; he was educated at Strasburg, acted for a time as ship's surgeon, and visited Spain and England. He then studied in Leyden and Paris, where he became interested in the discussion on the relative advantages of the different methods of cutting for the stone. Soon afterwards he settled in Amsterdam, where he taught anatomy privately and was appointed operating surgeon to the State Hospital. Finally, in 1713, he succeeded Bidloo as Professor of Medicine, Surgery, and Anatomy at the University of Leyden.

When Frère Jacques was operating in Holland Rau took the opportunity of dissecting the bodies of those who died, and thus learnt that the improved operation consisted in dividing the prostate and neck of the bladder. Knowing this and adopting it with much success he, deliberately and to his lasting shame, concealed the method and taught his pupils that he still cut through the posterior vesical wall, a dangerous operation with a high mortality. Amongst those who were thus taught was Albinus, Rau's favourite pupil and assistant. Albinus in turn taught Morand, the leading surgeon in Paris, and Cheselden in England performed the operation as it was described by Albinus. Both surgeons were profoundly dissatisfied with the results. They complained to Albinus, who, acting in perfect good faith, found that he had himself been deceived.

Cheselden (1688-1752), who was lithotomist to St. Thomas's Hospital from 1723 to 1727, immediately set himself to devise a more satisfactory operation based upon correct anatomical lines. After passing his operation through an experimental stage he thus described the technique:—

"I tie the patient as for the greater apparatus, but lay him upon a blanket several doubles upon an horizontal table about three feet high, with his head only raised. I first make as long an incision as I can cutting down between the musculus accelerator urine and erektor penis, and by the side of the intestinum rectum. I then feel for the staff, holding down the gut all the while

with one or two fingers of my left hand, and cut upon it in that part of the urethra which lies beyond the corpora cavernosa urethræ and in the prostate gland cutting from below upwards to avoid wounding the gut; and then passing the gorget very carefully in the groove of the staff into the bladder, bear the point of the gorget hard against the staff, observing all the while that they do not separate and let the gorget slip to the outside of the bladder. Then I pass the forceps into the right side of the bladder, the wound being on the left side of the perinæum; and as they pass, carefully attending to their entering the bladder, which is known by their overcoming a straitness which there will be in the place of the wound; then taking care to push them no farther, that the bladder may not be hurt I first feel for the stone with the end of them, which, having felt, I open the forceps and slide one blade underneath it and the other at top and then extract it very deliberately that it may not slip suddenly out of the forceps and that the parts of the wound may have time to stretch, taking great care not to gripe it so hard as to break it. And if I find the stone very large I again cut upon it as it is held in the forceps.

"Here I must take notice it is very convenient to have the bladder empty of urine before the operation; for if there is any quantity to flow out of the bladder at the passing of the gorget, the bladder does not contract but collapses into folds which makes it difficult to lay hold of the stone without hurting the bladder. But if the bladder is contracted it is so easy to lay hold of it that I have never been delayed one moment, unless the stone was very small. Lastly I tie the blood-vessels by the help of a crooked needle and use no other dressing than a little bit of lint besmeared with blood that it may not stick too long in the wound. And all the dressings during the cure are very slight, almost superficial, and without any bandage to retain them because that will be wetted with urine and gall the skin. At first I keep the patient very cool to prevent bleeding and sometimes apply a rag dipt in cold water to the wound and to the genital parts, which I have found very useful in hot weather particularly. In children it is often alone sufficient to stop the bleeding and always helpful in men.

"The day before the operation I give a purge to empty the guts and never neglect to give some laxative medicine or clyster a few days after if the belly is at all tense or if they have not a natural stool. What moved me to try this way, if I may be allowed to know my own mind, was the consideration of women scarce ever dying of this operation from which I concluded that if I could cut into the urethra beyond the corpora cavernosa urethræ the operation would be nearly as safe in men as in women.

"What success I have had in my private practice I have kept no account of because I had no intention to publish it, that not being sufficiently witnessed. Publicly in St. Thomas' Hospital I have cut two hundred and thirteen; of the first fifty only three died; of the second fifty, three; of the third fifty, eight; and of the last sixty-three, six. Several of these patients had the small-pox during their cure some of which died but I think not more in proportion than what usually die of that distemper; these are not reckoned among those who died of the operation. The reason why so few died in the two first fifties was that at that time, few very bad cases offered; in the third, the operation



being in high request, even the most aged and the most miserable cases expected to be saved by it. One of the three that died out of the one hundred and five was very ill with whooping cough; another bled to death by an artery into the bladder, it being very hot weather at that time. But this accident taught me afterwards, whenever a vessel bled that I could not find, to dilate the wound with a knife till I could see it.

"If I have any reputation in this way I have earned it dearly, for no one ever endured more anxiety and sickness before an operation, yet from the time I began to operate all uneasiness ceased and if I have had better success than some others I do not impute it to more knowledge but to the happiness of mind that was never ruffled or disconcerted and a hand that never trembled during any operation."

This is Cheselden's own account. Sauveur François Morand (1697-1773), son of the chief surgeon to the Hôtel des Invalides in Paris, was sent to England by the Royal Academy of France to report upon the operation in 1729, and states that he often saw a stone removed in twenty-four seconds and that it rarely took more than a minute. He watched Cheselden cut twenty-seven patients without losing one, and spoke so favourably of the method that it soon became known and practised throughout Europe. It remained in ordinary use until 1885, when lithotripsy—crushing the stone—came into vogue.

The *Transactions of the Royal Medico-Chirurgical Society* (1819, x, 153) contains an interesting personal account of the operation from the patient's side. It was performed on Dec. 30, 1811, and the anonymous patient says:—

"My habit and constitution being good it required little preparation of body, and my mind was made up. When all parties had arrived I retired to my room for a minute, bent my knee in silent adoration and submission and returning to the surgeons conducted them to the apartment in which the preparations had been made. The bandages &c. having been adjusted I was prepared to receive a shock of pain of extreme violence and so much had I over-rated it, that the first incision did not even make me wince although I had declared that it was not my intention to restrain such impulse, convinced that such effort of restraint could only lead to additional exhaustion. At subsequent moments, therefore I did cry out under the pain, but was allowed to have gone through the operation with great firmness.

"The forcing up of the staff prior to the introduction of the gorget gave me the first real pain, but this instantly subsided after the incision of the bladder was made, the rush of urine appeared to relieve it and soothe the wound.

"When the forceps was introduced the pain was again very considerable and every movement of the instrument in endeavouring to find the stone increased it. Still, however, my mind was firm and confident, and, although anxious, I was yet alive to what was going on. After several ineffectual attempts to grasp the stone I heard the operator say in the lowest whisper 'It is a little awkward, it lies under my hand. Give me the curved forceps' upon which he withdrew the others. Here, I think, I asked if there was anything wrong—or something to that purport—and was reanimated by the reply conveyed in the kindest manner, 'Be patient, Sir, it will soon be over'. When

the other forceps was introduced I had again to undergo the searching for the stone and heard Mr. Cline [1750-1827, Surgeon to St. Thomas's Hospital] say 'I have got it'. I had probably by this time conceived that the worst was over; but when the necessary force was applied to withdraw the stone the sensation was such as I cannot find words to describe. In addition to the positive pain there was something most peculiar in the feel. The bladder embraced the stone as firmly as the stone was itself grasped by the forceps; it seemed as if the whole organ was about to be torn out. The duration, however, of this really trying part of the operation was short and when the words 'Now, Sir, it is all over' struck my ear, the ejaculation of 'Thank God! Thank God!' was uttered with a fervency and fulness of heart which can only be conceived. I am quite unable to describe my sensations at the moment. There was a feeling of release, not from the pain of the operation, for that was gone and lost sight of, but from my enemy and tormentor with a lightness and buoyancy of spirits, elating my imagination to the belief that I was restored to perfect health as if by a miracle.

"I never heard what was the precise duration of the operation but conceive it to have been between twelve and fifteen minutes. With respect to the pain I am persuaded that if it were possible to concentrate what I have often suffered in one night into the same space of time it would have been less endurable. Indeed, this difference between the operation and one night's endurance of the stone was manifest. I have often been most distressingly reduced by the latter, but was not exhausted in the slightest degree by the former; at least my mind was firm throughout, and my body was not sensibly enfeebled. Upon the whole should I be again similarly afflicted, I should not hesitate in again submitting myself to the same mode of relief provided I could place myself in equally capable hands."

It is pleasant to know that this plucky English gentleman speedily recovered. He ceased to keep his bed at the end of the second week; was able to go downstairs at the end of the third; and walked nearly two miles by the end of the month. He rode on horseback, and was in fact perfectly cured at the end of the sixth week from the day of the operation; but the tone of the sphincter was but slowly regained, especially when walking or standing, and he then found it very difficult to retain his urine.

The portrait of Rau is copied from the illustration in Professor J. A. J. Barge's *De Leidse Anatomie*, Herdacht, 1923, p. 20.

THE INTERVERTEBRAL DISCS.

By N. ROSS SMITH, BOURNEMOUTH,

LATE SURGICAL REGISTRAR, ROYAL NATIONAL ORTHOPÆDIC AND WEST LONDON HOSPITALS.

THE objects of the research, the results of which are given in this paper, were to verify certain debatable points about the structure of the intervertebral discs, and to determine the mode of nutrition, the state of the discs from infancy to old age, and the diseases to which they are subject.

In the anatomy of the discs the following points were doubtful: The presence of a central synovial cavity; the nature of the cells in the nucleus pulposus; the size and position of the nucleus relative to the annulus; the relationship of the cartilaginous plates to the nucleus and annulus; and the presence of nerves, blood-vessels, and lymphatics. The function of the discs was not accurately known. The state of the discs in successive age-periods from birth to old age had not been systematically studied; nor had the pathological affections of the discs, other than destruction secondary to vertebral tuberculosis. There was thus a considerable amount of knowledge lacking concerning a group of structures which constitute a large proportion of the total length of the movable part and the largest ligamentous system of the spine. It was felt that the determination of this knowledge would aid in the interpretation of the clinical features of spinal affections.

The material for the anatomical and pathological investigation consisted of post-mortem specimens from 57 persons who died in a London Poor-Law hospital. The ages at death ranged from 1 year to 86 years. There were 2 subjects in the first decade of life, 4 in the second, 4 in the third, 6 in the fourth, 13 in the fifth, 10 in the sixth, 10 in the seventh, 7 in the eighth, and 1 in the ninth: 37 were males and 20 were females. Death had been due to a variety of acute and chronic diseases. No account of spinal symptoms was obtainable from the clinical records. One mid-thoracic and one lumbar disc were taken from each subject, permission to remove more not being obtainable.

In addition, with the assistance of Dr. W. H. Coldwell, the files of the X-ray Department of the Royal National Orthopædic Hospital for the years 1928-9 were searched for skiagrams of cases with spinal affections other than tuberculosis. From these and from private records a series of skiagrams showing affections of the discs was obtained.

OBSERVATIONS.

Anatomical.—The discs taken from each subject were sectioned horizontally and vertically and examined both macroscopically and microscopically. On macroscopic examination were noted the structure, shape, consistency, colour, compressibility, and elasticity. All these differed considerably in

different subjects; the observations made on them are described below under the headings of 'age changes' and 'pathological changes'. In the main, in the healthy discs of young subjects the observations are confirmatory of previous descriptions. It was noted that the size and position of the nucleus pulposus relative to the annulus fibrosus varied considerably; but as only two discs from each subject were examined, it cannot be stated whether or not this variation is according to any plan. In some specimens the nucleus was central, in others nearer the posterior or anterior margin; in some it formed a quarter of a horizontal section through the centre, while in others the proportion was as much as two-thirds. The line of demarcation between nucleus and annulus was sharp in healthy discs from young subjects, but ill-defined in all other discs.

On microscopic examination certain features were observed which have not been previously described. It was noted that the cartilaginous plates which bound a disc above and below are properly parts of the disc rather than



FIG. 212.—Cavity with villus in centre of the disc of a young person. ($\times 50$.)



FIG. 213.—High-power view of wall of cavity. ($\times 190$.)

of the vertebræ, as usually described. They consist of hyaline cartilage that is identical in structure with articular cartilage. One surface of each plate is applied to the cancellous tissue of the corpus vertebræ; on this surface bone-formation was seen to be taking place in the discs of subjects up to 18 years of age. The other surface is broken up into fibrillæ, which radiate into the tissue of the nucleus pulposus; between the fibrillæ, which are composed of hyaline ground substance, cells were seen transitional in character between cartilage cells and fibroblasts. At the margin of the plates there is gradual transition of the hyaline cartilage into the fibrocartilage of the annulus. The presence of the epiphysal centres of ossification in the periphery of the plates was noted in the discs of adolescent subjects.

Previous descriptions of the microscopic appearance of the annulus fibrosus—namely, obliquely running, parallel laminae of white fibrocartilage deeply, with a few superficial layers of ordinary fibrous tissue—were confirmed. In

the nucleus pulposus the striking feature of a central cavity was observed. In the disc of an infant (*Figs. 212, 213*) the extent of this space was seen to be almost as great as that of the nucleus, but the lumen was almost completely

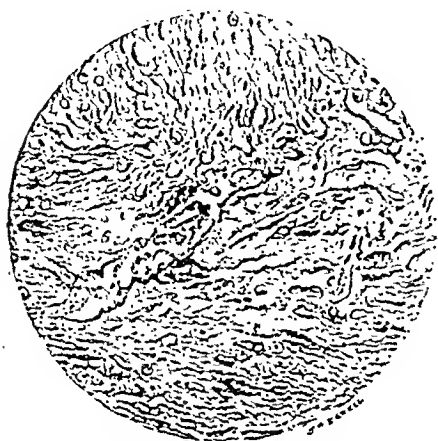


FIG. 214.—Nutritive channel containing leucocytes and coagulum in nucleus of a young healthy disc. ($\times 50$.)



FIG. 215.—Nutritive channels containing leucocytes in the annulus of a young healthy disc. ($\times 50$.)

filled by villous processes from the wall. In the discs of older subjects the size and form of the cavity varied considerably; in some in which pathological

changes were present a cavity was not seen. The tissue surrounding the cavity was composed mainly of a fine reticulum embedded in a gelatinous matrix. Numerous cells were present and were of three kinds: connective-tissue cells, fibroblasts, and cartilage cells. In the discs of young subjects fibroblasts and fibrous tissue were scanty; but at later ages they were plentiful. Cartilage cells existed singly or in groups of two or three and in varying proportions. Fibrillae of hyaline cartilage were present, particularly peripherally, where they radiated from the cartilaginous plates. The wall of the cavity was lined by a condensed layer, but there was no endothelium or continuous layer of cells as in synovial membrane. The



FIG. 216.—Nutritive channel containing leucocytes, erythrocytes, and coagulum in the cartilaginous plate of a young healthy disc. ($\times 50$.)

villi were more cellular than the wall of the cavity and were of irregular size and shape.

Nutritive channels were observed in sections from young healthy discs.

These were narrow spaces in the nucleus pulposus and annulus containing numerous leucocytes. In the nucleus they were seen in the midst of the reticular tissue, having a thin wall in which endothelial cells were not made out (*Fig. 214*). In the annulus they ran longitudinally between the fibres, and no wall was determinable. They could be traced to the periphery of the cartilaginous plates (*Fig. 215*), where they were clearly continuous with channels which pierced the plates from the marrow of the vertebral bodies (*Fig. 216*). Lining the channels through the plates was a single layer of flat elongated cells. Red blood-corpuscles were not seen in the channels in the nucleus and annulus, but were numerous in those through the plates.

Age Changes.—The macroscopic features of the specimens were as follows: In the first decade the disc was seen to be biconvex, highly compressible, and elastic, having a clear white annulus and a colourless, amorphous, gelatinous nucleus, sharply defined from the annulus (*Fig. 217*). The fibres of the annulus were slightly coarser in the lumbar than in the thoracic region. The cartilaginous plates were thin, translucent, and bluish-white. In the second decade the condition of the discs was similar, except that the fibres of the

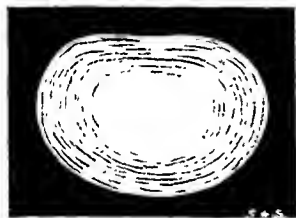


FIG. 217.—Horizontal section of a disc of an infant.

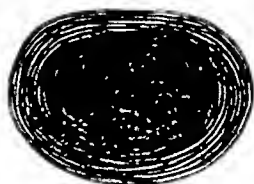


FIG. 218.—Horizontal section of a disc of an adolescent.

annulus were somewhat more coarse and a few fine fibres were present in the periphery of the nucleus (*Fig. 218*). In the third decade the fibres of the annulus were still fine, but the nucleus had become whitish, tougher, and fibrogelatinous, and the line of demarcation between annulus and nucleus less well defined. A few specimens exhibited impaired elasticity, moderate fibrosis, and yellow areas in the nucleus. In the fourth decade most of the specimens had diminished elasticity, moderately coarse fibres in the annulus, and a tough, white, fibrous, and amorphous nucleus. The nucleus passed without clear distinction into the annulus. Pathological appearances were more frequent than in the preceding decade. In the fifth decade pathological changes were common and severe. The fibres of the annulus were coarse, and the nucleus often appeared to be composed of whorls of fibrous tissue (*Fig. 219*). Few discs were more than slightly elastic. With loss of elasticity, the disc was often flattened or even biconcave, and the shape of the surfaces of the vertebral bodies was correspondingly altered. On section of the disc the nucleus did not bulge forward as in infancy, or was even retracted. In the sixth decade further advance was noted in the severity of the changes, the whole disc being inelastic and coarsely fibrous or

fibrocartilaginous (*Fig. 220*). The nucleus was often soft and pultaceous, or dry and friable; in colour it was grey or yellowish. Similar appearances were noted in the discs of subjects between 60 and 70 years. Some of the discs were markedly thinned and partly ossified peripherally; others were fibrous throughout and either not diminished in thickness or even slightly

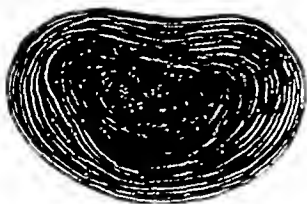


FIG. 219.—Horizontal section of a disc in the fifth decade, showing fibrous transformation.



FIG. 220.—Horizontal section of a disc in the sixth decade, showing fibrocartilaginous transformation.

increased. After the age of 70 these changes were accentuated still further (*Fig. 221*).

In the first two decades there were no specimens showing pathological changes, but in the third decade both the thoracic and lumbar discs of one subject showed marked fibrosis with yellow areas. In subsequent decades the

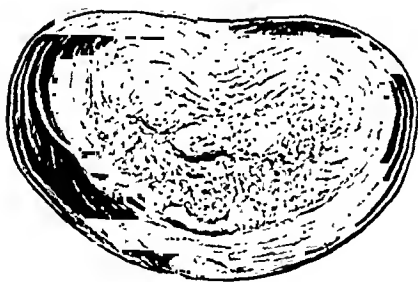


FIG. 221.—Horizontal section of a disc in the eighth decade, showing gross fibrocartilaginous and necrotic changes.

incidence of pathological changes progressively increased up to the age of 70, after which it decreased. In each adult age-period except the sixth and seventh, the specimens from one or more subjects showed surprisingly little difference in naked-eye appearance from specimens from young subjects; this was so as late as the eighth decade, in which the thoracic discs from two subjects had semi-gelatinous nuclei and high elasticity, although in one subject the lumbar discs were grossly changed.

The microscopic appearances observed in the discs of infants were

as described above. Sections from discs from older subjects showed cessation of calcification in the cartilaginous plates after the age of 18, gradual increase in the coarseness of the fibres of the annulus and in the proportion of fibrous tissue in the nucleus, increase in the number and size of groups of the cartilage cells both in the nucleus and annulus, and one or more forms of pathological change.

Tables I and II show the incidence and degree of changes in the discs in successive decades, and the sex incidence of the changes in the series of the research. The highest incidence is seen to be in the period 50 to 70 years,

with decline in the following decade. The changes were more frequent and severe among the male than the female subjects. They were as common and severe in the thoracic as in the lumbar discs, but the discs in the two regions were not always affected simultaneously or to the same degree; in some subjects the discs in one region were grossly changed, while those in the other region were little if at all altered.

Table I.—DEGREE OF DEGENERATION OF INTERVERTEBRAL DISCS IN SUCCESSIVE DECADES.

AGE	NIL		SLIGHT		MODERATE		SEVERE		TOTAL
	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	
Author's Series.—									
1-9	2	100.0	0	0.0	0	0.0	0	0.0	2
10-19	4	100.0	0	0.0	0	0.0	0	0.0	4
20-29	2	50.0	1	25.0	1	25.0	0	0.0	4
30-39	1	16.6	2	33.3	2	33.3	1	16.6	6
40-49	1	7.7	6	46.1	3	23.1	3	23.1	13
50-59	0	0.0	0	0.0	3	30.0	7	70.0	10
60-69	0	0.0	1	10.0	2	20.0	7	70.0	10
70-79	1	14.3	1	14.3	2	28.6	3	42.8	7
80-89	0	0.0	0	0.0	0	0.0	1	100.0	1
Norlen's Series.—									
18-30	15	100.0	0	0.0	0	0.0	0	0.0	15
30-40	15	100.0	0	0.0	0	0.0	0	0.0	15
40-50	23	92.0	2	8.0	0	0.0	0	0.0	25
50-60	28	56.0	20	40.0	1	2.0	1	2.0	50
60-70	30	36.6	40	48.8	11	13.4	1	1.2	82
70-80	17	17.0	49	49.0	32	32.0	3	3.0	100
80-89	5	22.7	7	31.8	9	40.9	1	4.6	22

Table II.—SEX INCIDENCE OF DEGENERATION OF THE INTERVERTEBRAL DISCS.

DEGREE OF CHANGE IN THE DISCS	MALE		FEMALE	
	Cases	Per Cent	Cases	Per Cent
Author's Series.—				
Nil	5	13.5	6	30.0
Slight	6	16.1	6	30.0
Moderate	9	24.5	5	25.0
Severe	17	45.9	3	15.0
	37	100	20	100
Norlen's Series.—				
Nil	81	48.2	59	36.0
Slight	61	36.3	62	37.8
Moderate	18	10.7	43	26.2
Severe	8	4.8	0	0.0
	168	100	164	100

Pathological Changes.—The changes in the discs from the condition in infancy are classifiable into fibrous, cartilaginous, calcareous, bony, fatty, necrotic, and liquefactive. The proportion of fibrous tissue in the discs and the degree of coarseness of the fibres were noted to increase gradually

in successive decades of life. Cartilage cells were seen in the nuclei of discs of infants, but increased in numbers and size of grouping rapidly in later years. It is difficult to say, therefore, whether the fibrous and cartilaginous transformations of discs observed are physiological or pathological. There were, however, in some discs from subjects as young as 20 years advanced fibrosis and cartilage-formation, whereas in some specimens from subjects in old age these changes were only moderately or slightly advanced. The relative amounts of fibrous tissue and cartilage were noted to vary. Some discs showed almost complete fibrous transformation, some about equal development of fibrous and cartilaginous tissue, and others great excess of cartilage over fibrous tissue. Changes were present in all parts of the disc, but chiefly in the nucleus. The original reticulo-gelatinous tissue of the nucleus was seen to persist in parts in some specimens even in the presence of advanced formation of fibrous tissue or cartilage. In the nucleus the fibrous tissue tended to assume a whorled form to

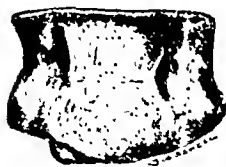


FIG. 222.—Showing ossification of the annulus and anterior longitudinal ligament.

naked-eye appearance. The cartilaginous hyperplasia took the form of clumps of several cells or of great masses of cells composing nodules.

Fat deposition was observed in many specimens. Necrosis with liquefaction was also common. Small groups of cartilage cells were often seen atrophied, while large nodules were usually necrotic centrally, and frequently disintegrated and liquefied: ragged cavities containing free cartilage cells and debris were thus formed. Complete bony transformation of a disc was not seen, nor was central calcification; but calcified nodules situated peripherally were seen in several specimens; in one there was ossification of the external layers of the annulus and of the anterior longitudinal ligament (*Fig. 222*).

The cartilaginous plates were noted to be often affected with the rest of the disc. Atrophy of the cells, which were surrounded by a zone of faintly stained matrix, and thinning of the plate was a common appearance. In some specimens fibrous transformation of the matrix was present. In others the cells were grouped into small clumps or even



FIG. 223.—Perforation and destruction of the cartilaginous plate of a disc by vascular connective tissue from the vertebral spongiosa. ($\times 50$.)

into large nodules, which extended through the whole thickness of the plate, and were continuous with nodules in the annulus or nucleus. In one grossly changed specimen strands of connective tissue containing arterioles were seen perforating and replacing the plates from the spongiosa of the vertebral body

and extending deeply into the centre of the disc (*Fig. 223*). This specimen was greatly thinned, and was composed of coarse fibrous and cartilaginous tissue, much of which was necrotic. The naked-eye appearance is shown in *Fig. 221*.



FIG. 224.—Normal appearance of the intervertebral spaces—sharply defined, biconvex, and completely translucent.



FIG. 225.—Thinning and flattening of the discs in a case of severe scoliosis with paraplegia in a young woman.
(*Author's case.*)



FIG. 226.—Peripheral calcification of the discs in a case of thoracic kyphosis in an old man.



FIG. 227.—Central calcification of a disc ('calcinosis intervertebralis') in an old woman: lateral view.

X-ray Appearances.—The following were noted, but no opportunity occurred to correlate them with histological observations: Thinning and flattening (*Fig. 225*); peripheral calcification (*Fig. 226*); central calcification

(*Figs. 227, 228*); obliteration by ossification (*Fig. 229*); diminished transparency with infection of the adjoining vertebræ (*Fig. 230*); thinning, flattening, and diminished transparency with osteochondritis of the adjoining vertebræ (*Figs. 231, 232*); abnormal biconvexity (excessive 'umbilication' of the vertebræ) with rarefying lesions of the vertebral body such as



FIG. 228.—Same case as *Fig. 227*: antero-posterior view.



FIG. 229.—Obliteration by ossification of a disc in a case of healed vertebral tuberculosis.



FIG. 230.—Diminished transparency of inflamed discs in typhoid spondylitis.

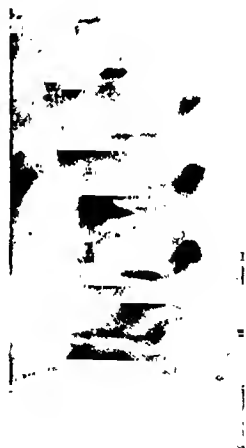


FIG. 231.—Early appearance of spinal osteochondritis. Thinning, flattening, and diminished transparency of the discs is present as well as fragmentation of the vertebral epiphysal plates.

osteoporosis (*Fig. 233*), carcinoma (*Fig. 234*), and compression fracture (*Fig. 235*). Thinning and flattening were noted in cases of scoliosis and kyphosis, sometimes alone, sometimes in the presence of osteo-arthritis or osteochondritis. Peripheral calcification was seen alone or with osteo-arthritis.

Complete ossification of a disc was observed in only one case—one of healed vertebral tuberculosis at the same site. The significance of the other appearances is discussed below.



FIG. 232.—Lateral appearance of spinal osteochondritis. The discs are thinned, flattened, and diminished in transparency, while the vertebral bodies are compressed and irregular.



FIG. 233.—Abnormal biconvexity of healthy discs with osteoporosis of the vertebræ. (Case of Dr. G. F. Still.)



FIG. 234.—Abnormally biconvex healthy discs with carcinoma of a vertebral body.



FIG. 235.—Abnormally biconvex healthy discs with compressed (post fracture) vertebral bodies.

In taking skiagrams of the discs it was found to be essential to take both lateral and antero-posterior views. In lateral exposures the axis of the spine must be parallel with the surface of the film. In judging the depth of the intervertebral spaces the varying direction of projection of the shadow due to the curves of the spine must be taken into consideration.

DISCUSSION.

The subject of the intervertebral discs has been much neglected, particularly by English workers. Apart from short descriptions in anatomical textbooks, there has been no writing on the subject in English since Cleland's paper in 1889.* In other languages there is to be found no writing between Luschka's monograph, published in 1858, and a few papers issued within the last ten years. As these writings are important and not readily available to English readers, it seems a service to include the substance of them in this discussion.

It is well known that the discs form approximately one-fourth of the total length of the movable part of the spinal column, and vary in vertical thickness in the several regions; they are thickest in the lumbar and thinnest in the upper part of the thoracic region. Recently, exact measurements have been made by Jacobi, whose material consisted of the spines of 102 persons ranging in age between 18 and 87 years. He found the smallest values for the discs in the upper thoracic region, where they averaged 3.9 mm., 5 mm. less than those for the lumbar region. Dividing his material into six groups according to age, and into four groups according to the state of the spine—normal, with discs degenerated but no other change, affected with spondylitis deformans, or osteoporotic—he found the proportion of the discs to the vertebrae approximately 1:5 in the thoracic and 1:3 in the lumbar region. He found further that in his series the proportions remained the same for all age-groups and in each of the four groups according to the state of the spine.

It is also known that the discs in the cervical and lumbar regions are thicker anteriorly than posteriorly and are largely responsible for the forwardly convex curves in those regions; in the thoracic region the discs are thinner anteriorly, in conformity with the posteriorly convex thoracic curve. These truths are important in understanding the effect of pathological changes in the discs upon the shape of the spine.

The discs are usually described as consisting of a peripheral fibrolaminar part (annulus fibrosus) and a central pulpy part (nucleus pulposus). Actually, as I have observed, there are three parts of the disc, distinct histologically and functionally, the third part being the thin plates of hyaline cartilage covering the upper and lower surfaces of the vertebral bodies. These plates are identical in composition with articular cartilage. Luschka measured their thickness and found it to be from 1 to 1.5 mm.

The oblique and cruciate arrangement of the fibres of the annulus is a striking feature, the reason for which has not been quite clear. Labat observed that the cruciate arrangement is indistinct in the cervical region, more evident in the thoracic, and well-marked in the lumbar. Rouvière has pointed out that the obliquity of the fibres is not the same at all levels of the spine. Measuring the fibres of the anterior part of the discs, he found that in the cervical and thoracic regions the fibres make with the vertical an angle of about 50°, whereas in the lower lumbar region the angle is at least 60°. These measurements are the same in the infant as in the adult. In the horse

* An article by Calvé and Galland, *Journal of Bone and Joint Surgery*, 1930, xii, No. 3, 555, has appeared since this paper was submitted for publication.

the figures for the cervical and lumbar regions are reversed. The peculiar arrangement of the fibres appears to be a provision for taking stresses in the movement of rotation around the vertical axis as well as in the infinite number of directions in which movement can be made around horizontal axes passing through the disc. An explanation, however, is required for the different obliquity of the fibres at different levels of the column, and for the greater obliquity in the lumbar region in man, where rotation is less than in the cervical region. Rouvière concluded that the inclination on the horizontal of the fibres is proportional to their length, which increases proportionately to the extent of the movements; but that the inclination also increases with the amount of superimposed pressure on the disc. Thus he explained why in the horse the obliquity of the fibres is greater in the mobile cervical region than in the less mobile lumbar region, whilst in man the reverse obtains because of the greater weight the lumbar discs have to bear.

The true nature of the structure of the nucleus pulposus, namely, synovial-like tissue enclosing and infolded into a cavity, was noted by Luschka. He stated that in the infant the cavity is large, occupying more than half the intervertebral space, though filled with villi; in the aged the cavity is commonly narrow, but may be wide (*Figs. 236, 237*). I have been able to confirm these statements by microscopic examination, but not macroscopically. It is clear that an intervertebral disc must be considered, as Luschka stated, a rudimentary diarthrodial joint, in which the cartilaginous plates represent the articular cartilages; the annulus, the fibrous capsule; and the nucleus, the synovial membrane and cavity. In the development of a disc the synovial membrane does not disappear over the articular cartilages, as it does in fully developed diarthrodial joints.

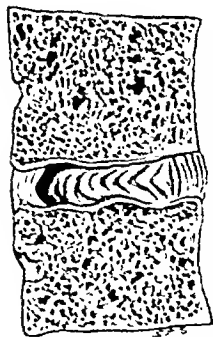


FIG. 237.—Vertical section to show the same feature as Fig. 236. (*Luschka.*)

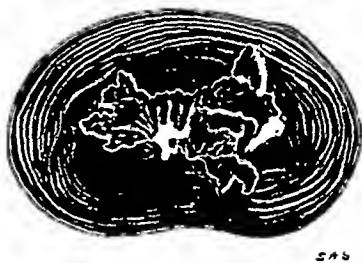


FIG. 236.—Horizontal section (dissected) of an intervertebral disc, showing the synovial-like cavity. (*Luschka.*)

The function of an intervertebral disc does not seem to depend on its arrangement as a joint, but upon the physical difference between its constituents and the formation of the fibres of the annulus. The nucleus pulposus is normally highly compressible and elastic, whereas the annulus is relatively inelastic: a disc thus forms a delicate buffer between the vertebrae, absorbing shocks transmitted through the spine. The fibres of the annulus are strong ligaments between the vertebrae, but by their peculiar arrangement allow a small range of movement in an infinite number of directions. According to Schmorl the position of the nucleus varies in different parts of the spine, so that the weight above is distributed evenly over each disc. I have not been able

to examine a sufficient number of discs in any subject to verify this, though I have noted that the size and position of the nucleus varied considerably in my specimens.

The observation of the presence of nutritive channels in the disc is in conflict with the commonly held view that nourishment is provided by diffusion currents from the spongiosa of the vertebræ. On the other hand, it is confirmatory of the conception of the discs as diarthrodial joints. The appearances in my sections establish that nutritive channels exist; they come from the marrow of the vertebral bodies and reach the centre of the disc by first piercing the cartilaginous plates and then running between the fibres of the annulus. I regard them as blood-vessels, but I have not had the opportunity of confirming my observations by injection methods.

Age Changes.—The observations I have made are similar to those of Luschka and Norlen. Luschka described only the appearances of the discs in infancy and old age, and attributed the change to senility. Norlen's investigation was of specimens from 309 subjects between the ages of 18 and 89 years. His observations differ from mine in the following respects: greater frequency of change in females than in males; absence of naked-eye changes before the age of 40 years; and greater incidence and severity of changes in the lumbar than in the thoracic discs (*see Tables I, II*). In both series the nucleus pulposus is clearly the chief seat of change and there is falling-off of the incidence of naked-eye appearances of change in the last as compared with the middle decades of life. The widely varying age of onset and their nature suggest that the changes are not attributable to age alone. Whatever the cause, it is clear that the increasing density and impairment of resiliency of the discs must be responsible for much of the loss of suppleness of the trunk which is usual after the age of 30. Individuals who retain their youthful suppleness after that age do so because they have suffered no changes of their intervertebral discs.

Pathological Changes.—These are divisible into degenerative and inflammatory. Though the fatty, necrotic, and liquefactive changes appear to be degenerative, it is uncertain whether the fibrous, cartilaginous, calcareous, and bony changes are degenerative, inflammatory, or physiological. On the whole, there is gradual increase in the proportion of fibrous and cartilaginous tissue in successive decades of life, and the changes are similar to those of other mesenchymal tissues with advancing years. But fibrous and cartilaginous formations have been seen advanced in subjects as young as 20 years, and have been present in only slight degree in aged subjects. Lyon considers that these changes may occur prematurely through the strain of heavy bodily labour or through the influence of general diseases: in the metabolic disorder alkaptonuria, ochronosis (blackening) of the discs may be present and predispose to degeneration from injury. This is interesting in view of Garrod's observations on the peculiar liability of subjects of alkaptonuria to ochronosis of the articular cartilages of the diarthrodial joints followed by osteo-arthritis, in which, as is remarked below, the pathological appearances are not unlike those seen in the discs. Moreover, Garrod comments on the conspicuousness of the spinal lesions in alkaptonuria. Among other possible causes of the changes may be mentioned contusion and rupture. According to Beneke,

rupture of a disc is by no means rare, resulting in diffuse degeneration at and around the site of rupture. Lyon quotes Schmorl's observation of two cases of laceration of the discs between the 4th and 5th lumbar vertebræ during operations performed in the Trendelenburg position. He also mentions Hoffmann's experiments on the effect of weight-traction on isolated columns of thoracic and lumbar vertebræ, in which it was found that rupture took place always through the lowest disc, the posterior longitudinal ligament being the first point to give way.

Calcification of the centre of the disc has been noted radiologically by many observers since it was originally described by Calvé and Galland in 1922. None of my specimens shows this change, and I have not found it described pathologically by others. In a skiagram a shadow is seen in the centre of one or more intervertebral spaces. This is sometimes single, sometimes partite (*see Figs. 227, 228*). Most often it has been seen in the thoracic and lumbar regions of the spines of persons in middle or later life and has been discovered accidentally. In these cases the appearance seems to have been evidence of degeneration; but in other cases it has been thought to be the result of an inflammation. Baron's case was a child of 12 years; on the eighteenth day of an acute illness with severe pain in the back, lateral curvature with muscular rigidity and increased tendon-reflexes, a skiagram showed a dense transverse shadow between the 12th thoracic and 1st lumbar, and between the 1st and 2nd lumbar vertebræ. These shadows persisted for months, but after a year were no longer demonstrable. The symptoms disappeared after six months with treatment by a corset. Baron assumed that the shadows were due to rapid deposition of calcium in the centre of the discs because of special affinity for calcium of inflamed cartilaginous tissue. Giongo's case was a tuberculous subject, age 44 years, in whom shadows were discovered accidentally in two intervertebral spaces in the thoraco-lumbar region during the course of an examination of the alimentary tract. Giongo thought, therefore, that tuberculosis played a part in the process in the discs. Buffnoir described five cases with slight kyphosis and severe pains in the back radiating to the legs, which ran an intermittent course; dense shadows in the intervertebral spaces were visible in skiagrams. Lyon, on the basis of recent investigations by Schmorl, maintains that in Baron's case the lesion of the disc was secondary to one in the corpus vertebræ; he describes the case as 'spondylitis gripposa with secondary involvement of the discs'. Baron gave the name 'chondritis intervertebralis acuta'. Other writers use the term 'calcinosis intervertebralis' to denote, generally, a calcified area in the disc.

The effect of infectious diseases on the discs has been studied by Schmorl. X-ray examination of vertebral columns affected by infectious diseases, particularly typhoid fever, revealed diminished transparency and thickness, and later, obliteration of the intervertebral spaces. Nearly always the lumbar region was the one affected. At an early stage, when the spongiosa of the vertebræ has become inflamed, the normal elasticity of the as yet unaffected discs exerts pressure which causes collapse of the diseased parts of the adjoining vertebræ. Later, the discs are themselves inflamed by extension from the vertebræ, vascularized, and finally shrink from fibrotic and degenerative

changes, or become ossified from bone-forming tissue which enters with the vessels. A case is described by Rugh in which in place of the discs between the 3rd and 4th lumbar vertebræ dense vascular connective tissue was found passing into the vertebral bodies. Lyon maintains that inflammation of a disc in infectious disease is secondary to an affection of the corpus vertebræ, and probably never primary. According to the stage at which the process is arrested, there remain excessive unibilication of the vertebræ, narrowing or obliteration of the intervertebral space.

RELATIONSHIP OF AFFECTIONS OF THE DISCS TO OTHER DISEASES.

Arteriosclerosis.—The possible relationship between arteriosclerosis and degeneration of the discs was studied by Norlen. He noted that in his series both affections were chiefly found in old subjects, but their relative incidence in a charted record did not run quite parallel (*Fig. 238*). The

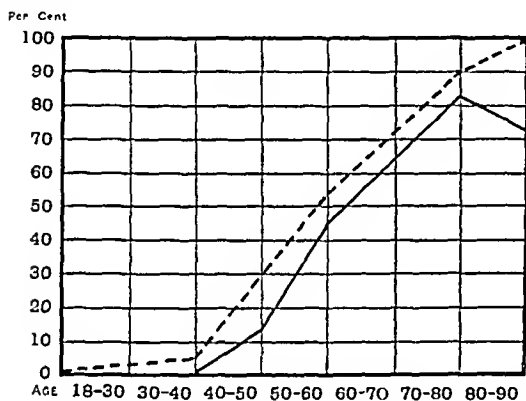


FIG. 238.—Relative incidence of arteriosclerosis and degeneration of the intervertebral discs. The continuous line shows the percentage of cases with degenerated discs in the various decades, and the dotted line the percentage of cases with arteriosclerosis in the same groups. (Norlen.)

difference was slight, the curve of both rising continuously as age advanced, until old age, when it became marked. Arteriosclerosis was found in 100 per cent of persons between 80 and 90 years of age, whilst degeneration of the discs was absent in 20 per cent. Norlen thought also that probably the two processes are not concurrent in the earlier decades, there being no constant evidence of simultaneous onset: indeed, arteriosclerosis appeared to begin at an earlier age. In their more severe forms the two states seemed to occur in direct opposition.

Osteoporosis.—Norlen also investigated the possible inter-relationship of osteoporosis of the vertebræ and degeneration of the discs. In this, as with arteriosclerosis, his observations were unfinished; but they are sufficiently striking to warrant further investigation. Of 21 subjects showing osteoporosis, he noted that in 8 there were no macroscopic signs of degeneration in the discs, 10 showed only slight changes, and 3 moderately advanced changes. Thus it appeared that osteoporosis of the vertebræ and degeneration of the discs do not arise simultaneously or proceed concurrently. Norlen's impression was that they run in inverse proportion to one another. When the discs degenerate they become flattened or biconcave instead of biconvex as normally. In severe osteoporosis the concavity of the vertebral surfaces is increased, the discs often remaining biconvex (*Fig. 239*). Hence it seems that the etiological factors differ for

the two states, and that some reciprocal adaptability exists between the vertebrae and the discs when either is diseased. Norlen noted the same phenomenon in destructive processes, such as cancer metastasis, in the vertebrae, the discs in the affected region being markedly healthy and elastic. These observations are confirmed by those of Schmorl, who maintains that the healthy discs actually aid by pressure the destructive process in the vertebrae; he observed that spinal curvature with osteoporosis is most severe when the discs are intact.

The radiographic appearance of the condition of healthy discs with osteoporosis of the vertebral bodies is excessive umbilication of the upper and lower surfaces of the vertebrae (*see Fig. 233*). Umbilication is necessarily a normal skiagraphic feature because of the biconvex shape of the discs. Exaggeration of this feature may not be significant of disease, since it may be present with lordosis, when presumably the nuclei of the discs are pressed forward and thus exert abnormal pressure on the vertebral bodies.

Osteochondritis of the Spine.— In this condition, which has been recognized recently, the X-ray appearances make it clear that the discs are as much affected as the vertebrae. In cases of recent onset the appearance of diminished thickness and transparency of the discs is as prominent a feature as frag-

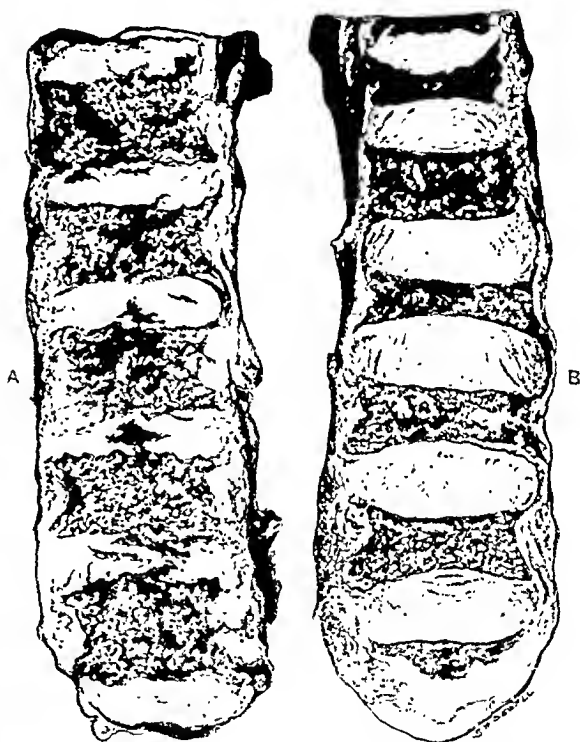


FIG. 239.—Coronal sections of two vertebral columns. A, Degenerated discs with only slightly osteoporotic vertebrae. B, Healthy discs with severely osteoporotic vertebrae. (Norlen.)

mentation, blurring, and irregularity of the margins of the vertebrae (*see Fig. 231*). In late cases thinning of the discs is as apparent as diminished thickness, antero-posterior wedging, and increased density of the upper and lower margins of the bodies of the vertebrae (*see Fig. 232*). The disease is an affection of the periods of rapid growth in childhood and adolescence, and has its greatest incidence in the thoracic region. Usually several vertebrae and the intervening discs are affected. The symptoms are pain in the affected region and rounded kyphosis or kypho-scoliosis; muscular spasm and local tenderness are present in some cases. The etiology and pathology are conjectural. None of my specimens came from a subject known to have had the disease, and I can find no record of histological examination by others. On

analogy with osteochondritis of other sites it is generally assumed that the affection is inflammatory and infectious.

Osteo- and Rheumatoid Arthritis.—In studying the changes in the intervertebral discs one cannot fail to be struck by the resemblance of these to the changes in joints in chronic rheumatic arthritis. Atrophy and vascularization of the articular cartilages, fibrosis and deposition of fat in the synovial tissue, and thickening and ossification of the capsule are characteristic of the rheumatoid type of chronic arthritis, and are the appearances in many specimens of intervertebral discs. In other specimens formation of cartilaginous nodules, which may calcify, centrally and peripherally, as well as fibrosis of the nucleus and annulus, give a picture similar to that of the osteoarthritic type. Moreover, the age of maximum incidence of degeneration of discs is the same as of chronic arthritis, and the affections are often present simultaneously and affect chiefly the same regions of the spine. Indeed, assuming, as is believed by many, that traumatism is a causative factor in both, degeneration of the discs can scarcely be present to any degree without leading to chronic arthritis of the spine, and vice versa. When the discs degenerate and lose their elasticity, the bones and joints of the spine are abnormally subjected to the strain of the body weight and to shocks transmitted through them undiminished by the buffer action of the discs. On the other hand, when the spine is limited in movement or deformed by osteoarthritis or other cause, the intervertebral discs probably suffer minor injuries which result in fibrosis or other change. Manifestly, in a proportion of cases, pain and stiffness of the back attributed to rheumatic affections of the muscles, bones, or joints of the spine are due partly or solely to changes in the discs; also to no small extent deformity from rheumatic arthritis—as well as scoliosis and other chronic affections of the spine—is often due to alteration in the shape and elasticity of the discs.

CONCLUSIONS.

1. The intervertebral discs are rudimentary diarthrodial joints possessing a cavity filled with villi, a fibrocartilaginous capsule, and upper and lower cartilaginous plates comparable to articular cartilages.

2. The mode of nutrition of the discs is by blood-channels which pierce the cartilaginous plates from the marrow of the vertebral bodies.

3. The discs allow a small degree of movement in all directions between the vertebræ, and thus are highly important in imparting flexibility to the spine. They are also powerful buffers between the vertebræ, absorbing shocks transmitted along the spine.

4. In the first two decades of life an intervertebral disc is biconvex and highly elastic; the fibres of the annulus are fine and white, the cartilaginous plates thin, translucent and bluish-white, and the nucleus amorphous, gelatinous, colourless, and sharply defined. The nucleus varies in size and position relative to the annulus. In subsequent decades the disc tends to become progressively more coarse, inelastic, tough, and discoloured. In some individuals the discs preserve their juvenile state until late in life.

5. The lesions of the discs which have up till now been described are

fibrous, cartilaginous, calcareous, bony, fatty, necrotic, and liquefactive changes. A special form which has been noted radiologically is calcification of the centre of the disc, to which the term 'calcinosis intervertebralis' has been applied. Inflammatory lesions have been observed in influenza and typhoid fever. Inflammatory lesions are thought to be secondary to an affection of the spongiosa of the corpus vertebræ. In osteochondritis of the spine the discs are affected as well as the vertebræ.

6. A relationship appears to exist between affections of the disc and other diseases—notably arteriosclerosis, osteoporosis, osteochondritis, and chronic rheumatic arthritis. There seems also to be some reciprocal adaptability between the discs and the vertebræ when either are diseased.

7. (a) The usual progressive loss of suppleness of the body after the age of 30 years is due in great part to the increased density and diminished resiliency of the intervertebral discs. (b) In a proportion of cases back pain and stiffness, usually attributed to rheumatic affections of other tissues of the back, are due to affections of the discs. (c) The symptoms in osteoarthritis and other chronic affections of the spine are often due in part to changes in the discs. (d) The discs when healthy accentuate the effects of rarefying lesions of the vertebral bodies.

8. Diagnosis of lesions of the intervertebral discs depends on radiographic examination. A lateral view with the axis of the spine parallel to the surface of the film is essential, as well as an antero-posterior view. Thinning and flattening, diminished transparency, peripheral or central calcification, obliteration by ossification, or abnormal biconvexity may be observed.

I am indebted to Sir Arthur Keith for suggesting the subject of this research and for criticism and advice; also to him and to Mr. Lawford Knaggs for help in the interpretation of histological features. Dr. W. H. Coldwell has aided me greatly in the collection of the skiagrams, most of which were taken by him; two are by Dr. Leiba Buckley. The work was carried out with the aid of a Research Grant from the British Medical Association, to which I wish to express my gratitude.

BIBLIOGRAPHY.

- BARON, *Jahrb. f. Kinderheilk.*, 1924, civ, 357.
 BENKE, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1925, xxxiii, 844.
 BURNOR, *Bull. med.*, 1923, No. 10 (quoted by Lyon).
 CALVÉ, *Jour. de Radiol. et d'Electrol.*, 1925, ix, 22.
 CALVÉ and GALLAND, *Ibid.*, 1922, vi, 21.
 CLELAND, *Jour. Anat. and Physiol.*, 1889-90, xxiv, 373.
 GARROW, *Proc. Roy. Soc. Med.*, 1923, xvii, 2.
 GIOINGO, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1928, xxxvii, 873.
 HOFMANN, *Arch. f. klin. Chir.*, 1925, cxxxv, 412.
 JACOBI, *Beitr. z. pathol. Anat.*, 1927, lxxviii, 303.
 LABALT, *Med. Gazette*, 1835-6, xvii, 341.
 LUSCHKA, *Arch. f. pathol. Anat.*, 1855, ix, 312; *Die Halbegelenke d. menschlichen Körpers*, 1858, Berlin.
 LYON, *Arch. f. Orthop.*, 1928, xxvi, 295.
 NORLÉN, *Hygiea*, 1927, lxxxix, 159.
 ROUVIERE, *Comptes rend. Soc. de Biol.*, 1921, lxxxv, 156.
 RUGH, *Amer. Jour. Orthop. Surg.*, 1915, xiii, 289.
 SCHMOLZ, *Orthop. Gesellsch. Kong.*, 1927, xxi, 3.

THE MECHANISM OF DEEP TENDERNESS IN GASTRIC AND DUODENAL ULCER.

By JOHN MORLEY,

ASSISTANT SURGEON AT MANCHESTER ROYAL INFIRMARY;

AND E. W. TWINING,

ASSISTANT RADIOLOGIST AT MANCHESTER ROYAL INFIRMARY.

IN any attempt to elucidate the difficult problem of pain due to lesions of the stomach and duodenum it is essential to distinguish quite clearly between spontaneous visceral pain on the one hand, and objective tenderness on the other. The stomach, as is well known from the work of Mackenzie and Lennander, verified since then by many observers, is quite insensitive to the ordinary mechanical, chemical, or thermal stimuli which cause pain when applied to the sensory somatic nerves. This fundamental fact has caused all the difficulty in explaining the mechanism of visceral gastric pain. We take the existence of true visceral gastric pain as proved, in spite of the teaching of Mackenzie, who regarded it as referred to the nerves of the abdominal parietes. With the question of the precise stimulus adequate to produce it we are not concerned in this paper. Whether it is due to increased intragastric tension (Hurst), to stimulation by hydrochloric acid (Palmer), to vascular congestion in the region of the ulcer (Kinsella), or to some other stimulus as yet unrecognized, is a problem entirely apart from the one with which this investigation deals. Spontaneous visceral pain in gastric and duodenal ulcer sometimes occurs without any tenderness on pressure, and tenderness on pressure may be found apart from any spontaneous pain. The visceral pain is felt in the centre of the epigastrium, but is not localized with any precision by the patient, and does not move with changes of position of the stomach, whereas deep tenderness, as we shall see, is very accurately localized.

CONFLICTING THEORIES ON THE MECHANISM OF DEEP TENDERNESS.

Mackenzie regarded both the spontaneous pain and the objective tenderness of gastric and duodenal ulcers as 'referred' by what he called a viscerosensory reflex, through the afferent splanchnic nerves and the spinal cord to the parietes. This theory of his led him to minimize the distinction between spontaneous pain and tenderness, and to look for both spontaneous pain and tenderness in the same place. He refers to "the site of pain and hyperalgesia" as though they invariably coincided. It also led him to the belief that the "site of pain and tenderness" was usually remote from the position of the ulcer. In support of this view he quotes several cases in which operative or post-mortem findings appeared to support this contention. It must be remembered, however, that when the abdomen is opened at operation, and still more at post-mortem examination, the relation of the

stomach to the abdominal wall is by no means the natural one. The only method of examination free from this fallacy is that of visualizing the ulcer crater by X rays, and when Mackenzie made his observations radiological technique had not advanced sufficiently to give him this aid.

Hurst, in his Goulstonian Lectures on the sensibility of the alimentary canal (1911), while he rejected Mackenzie's views as to visceral pain, accepted that part of Mackenzie's theory which attributed the objective tenderness to a viscerosensory reflex. He states: "Pressure applied over a gastric ulcer could not possibly cause local pain, as ulcers in the alimentary canal are insensitive to mechanical stimuli. In the large majority of cases the epigastric tenderness is a result of the irritable focus in the spinal cord produced by abnormal impulses reaching it from the stomach."² Like Mackenzie, Hurst fully admits that where the disease process in gastric ulcer extends to the parietal peritoneum, both localized tenderness and muscular rigidity are produced: but these authorities evidently regarded this as an exceptional process, and believed that the common manifestations of deep tenderness in gastric and duodenal ulcer are referred through the agency of the afferent splanchnic nerves and spinal cord to the abdominal wall.

Hurst's Later Theory.—In his latest writing on the subject³ Hurst takes an entirely new view of visceral tenderness. He subdivides the section on tenderness in gastric and duodenal ulcer into: (1) Visceral tenderness; and (2) Reflex tenderness and rigidity. Under the heading of 'visceral tenderness' he describes a localized deep tenderness which corresponds accurately with the position of the ulcer as visualized by X rays, and shifts with the ulcer in different postures. He concludes that this phenomenon, which had not been observed at the time of his Goulstonian Lectures, can only be interpreted as meaning that the subserous layer of the *visceral* peritoneum over an ulcer is sensitive to pressure. Our grounds for dissenting from this conclusion will be set forth later in this paper. In dealing with what he describes as 'reflex tenderness and rigidity', Hurst still accepts the well-known theories of Mackenzie.

THE SCOPE OF THIS INQUIRY.

It appeared to us necessary in the first place to investigate the relationship between localized points of deep tenderness and the actual lesion in a series of cases of gastric and duodenal ulcer. We hoped by so doing to ascertain whether the point of maximum deep tenderness as determined by palpation does in fact correspond with the position of the underlying ulcer, as Hurst and also Kinsella⁴ contend, or whether, as Mackenzie held, they do not coincide. In this way, it seemed to us, we should obtain data which would show whether the facts are consistent with Mackenzie's theory of referred tenderness, or with Hurst's later theory of direct visceral tenderness, or with both theories or neither.

METHOD OF INVESTIGATION.

Only patients who had both a demonstrable ulcer and a localized area of deep tenderness were investigated. In each case the site of the point of

deep tenderness was charted in the ordinary clinical examination: (1) In the supine position; (2) In a lateral position, left or right; and (3) When standing erect. As a rule the stomach was empty when this was done. The stomach was then visualized by barium under the fluorescent screen in the erect position, the point of maximum deep tenderness was marked by a metallic ring, and a radiogram taken. The patient was then placed in the supine position with the ring left *in situ*, the point of maximum tenderness found in this position marked by a second ring, and another film exposed. It was not found practicable to take satisfactory radiograms in the lateral positions. The charts accompanying the case reports given below show the points of maximum deep tenderness in the various positions.

CASE REPORTS.

Case 1.—A. L., female. Gastric ulcer.

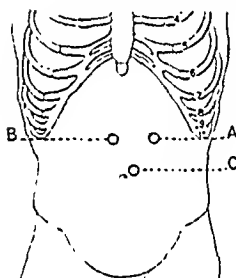


FIG. 240. — *Case 1.* Gastric ulcer. A, Supine; B, Right lateral; C, Erect.

Well localized point of deep tenderness in left of epigastrium in supine position (*Fig. 240A*). When lying on her right side there was a marked shift of the tender point to the right of the mid-line (*Fig. 240B*). In the erect posture the tender point moved downwards 2 in. (*Fig. 240C*).

X RAY.—Erect: ring placed on point of deep tenderness was found to correspond with the visualized crater (*Fig. 241*). Supine: a second ring placed on the new position of the tender point also corresponded with the crater (*Fig. 242*).

OPERATION.—Large ulcer on the lesser curvature and the posterior wall of stomach, with a patch of anæmic tissue on the anterior aspect near the lesser curvature that looked like perforating. Marked induration round ulcer, which was adherent posteriorly. Schoemaker gastrectomy.

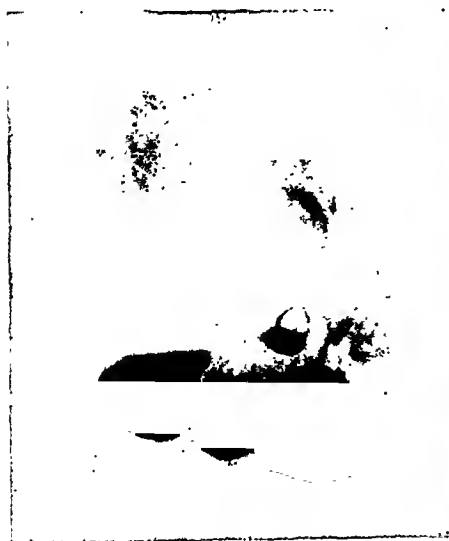


FIG. 241.—*Case 1.* Erect position.

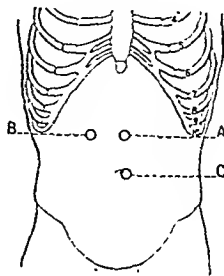


FIG. 242.—*Case 1.* Supine position.

Case 2.—G. B. S., male. Gastric ulcer.

This patient had had a perforated ulcer sutured in another hospital seven years before. He now had symptoms of gastric ulcer. Localized point of deep tenderness to left of the mid-line in the epigastrium (*Fig. 243A*). The tender point shifted to the right in the right lateral position (*Fig. 243B*), and downward in the erect posture (*Fig. 243C*).

FIG. 243.—*Case 2.* Gastric ulcer.
A, Supine; B, Right lateral; C, Erect.



X RAY.—Erect: the tender point, a little to the left of the umbilicus, corresponded with a visible crater on lesser curvature (*Fig. 244*). Supine: the tender point was higher and again corresponded with the visualized crater (*Fig. 245*).

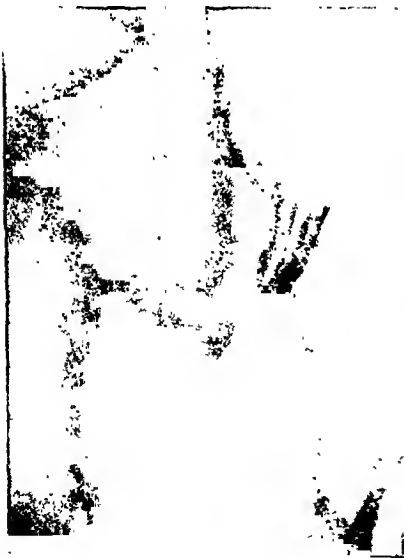


FIG. 244.—*Case 2.* Erect position.



FIG. 245.—*Case 2.* Supine position.

OPERATION.—Small ulcer high up on the lesser curvature, not adherent. Small scar of old ulcer on the anterior wall of first part of the duodenum, now healed. Schoemaker gastrectomy.

Case 3.—W. L., female. Duodenal ulcer.

Well localized point of deep tenderness, shifting in the left lateral (*Fig. 246b*) and erect positions (*Fig. 246c*) as compared with the supine (*Fig. 246a*).

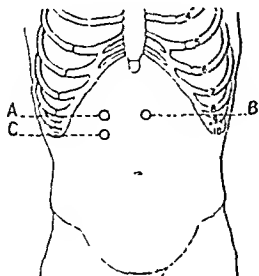


Fig. 246.—*Case 3.* Duodenal ulcer.
A, Supine; B, Left lateral; C, Erect.

X RAY.—Showed that the point of deep tenderness in both erect and supine positions corresponded with the duodenal cap (*Figs. 247, 248*).

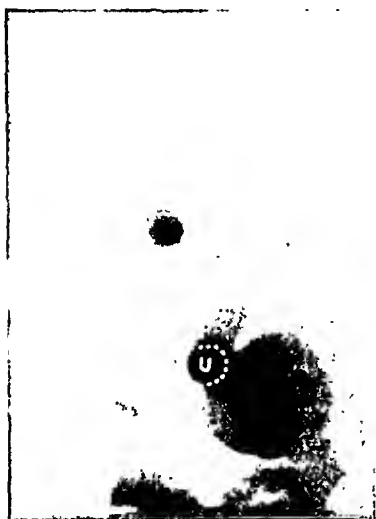


Fig. 247.—*Case 3.* Erect position.
U, Umbilicus.

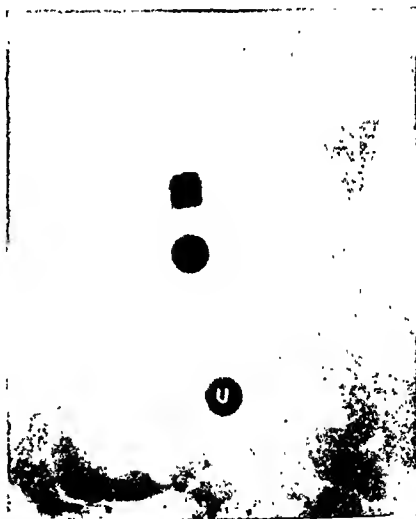


Fig. 248.—*Case 3.* Supine position.
U, Umbilicus.

OPERATION.—Well-marked ulcers on the anterior and posterior walls of the first part of the duodenum. Stippling of serous coat of the anterior wall on handling. No fibrosis over ulcer. Posterior gastro-enterostomy.

DEEP TENDERNESS IN PEPTIC ULCER 381

Case 4.—J. H., male. Chronic gastric ulcer.

No cutaneous hyperalgesia. The deep tenderness was more diffuse than usual (*Fig. 249*), but the difference between its position in the erect and the supine posture was marked. No lateral shift made out when the patient lay on his right side.

X RAY.—The point of deep tenderness corresponded in both positions with the crater on the lesser curvature near the pylorus. Vertical shift of crater and tender point 2 in.

OPERATION.—Large saddle-shaped ulcer on the lesser curvature just above the pylorus, adherent to the liver and pancreas. On the anterior surface of the stomach over the ulcer was a shaggy mass of organized fibrous tissue over an area 2 in. in diameter exactly corresponding to A in *Fig. 249*. Posterior gastro-enterostomy.

Note.—The wide roughened area of stomach wall in contact with the anterior parietal peritoneum should be noted. We believe it to explain the unusually extensive area of localized deep tenderness. The significance of this will be discussed later.

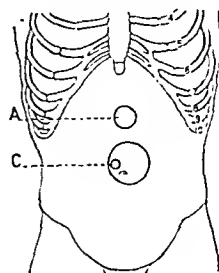


FIG. 249.—*Case 4.* Gastric ulcer. A, Supine; C, Erect.

Case 5.—E. H., male. Gastric ulcer.

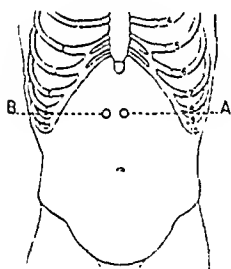


FIG. 250.—*Case 5.* Gastric ulcer. A, Supine; B, Right lateral. No downward shift in erect position.

Point of deep tenderness in centre of the epigastrium (*Fig. 250A*). Slight shift to the right when in the right lateral position (*Fig. 250B*). No downward shift in the erect posture.

X RAY.—The point of deep tenderness corresponded with the ulcer high on the lesser curvature. The point was first marked after a few mouthfuls of barium had been given. When the stomach was completely filled the tender point was 1 in. lower, and in both positions corresponded with the crater. In the supine position the tender point was lower than the visualized crater (? residual tenderness with actual crater now out of reach).

OPERATION.—Small ulcer on lesser curvature. Schœmaker gastrectomy.

Case 6.—L. G., female. Duodenal ulcer.

Localized tender point with right lateral and vertical shift (*Fig. 251*).

X RAY.—Supine: There was very well defined tenderness to the right of the lesser curvature, between it and the duodenal cap. Erect: it appeared to be nearer the lesser curvature. It was also found to be lower when the stomach was filled with barium than it had been after the first few mouthfuls. When the patient was again examined in the supine position the tender point was lower than it had been with the stomach empty.

OPERATION.—Well-marked ulcer on the first part of the anterior wall of the duodenum just beyond pyloric ring. No lesion on lesser curvature. A very mobile stomach and duodenum, and the duodenum particularly showed a wide range of mobility. Schœmaker gastrectomy.

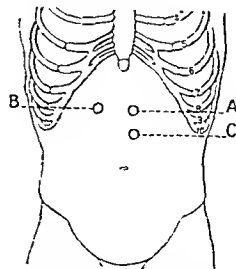


FIG. 251.—*Case 6.* Duodenal ulcer. A, Supine; B, Right lateral; C, Erect.

Case 7.—A. G., male. Gastric ulcer.

Localized tender point showing typical shift in the right lateral and erect postures (*Fig. 252*).

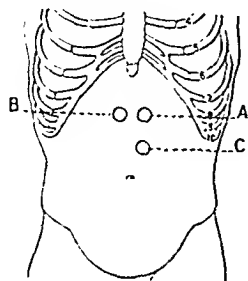


FIG. 252.—*Case 7.* Gastric ulcer.
A, Supine ; B, Right lateral ; C, Erect.

X RAY.—The tender point corresponded accurately with the crater in both positions (*Figs. 253, 254*).



FIG. 253.—*Case 7.* Erect position.



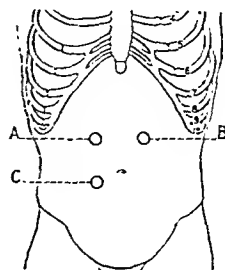
FIG. 254.—*Case 7.* Supine position.

OPERATION.—Ulcer on the lesser curvature 2 in. above the pyloric ring. Marked fibrosis round it, and petechial stippling on sponging. Schoemaker gastrectomy.

Case 8.—J. C., male. Chronic duodenal ulcer with pyloric obstruction.

Tender point to the right of the mid-line, with usual left lateral and vertical shift (*Fig. 255*).

FIG. 255.—*Case 8.* Duodenal ulcer.
A, Supine; B, Left lateral; C, Erect.



X RAY.—Stomach full of fluid, and pylorus and duodenal cap not satisfactorily defined. A trace of barium in the cap showed its position. The tender points, with large vertical shift, lay a little to the left of those found before giving barium, and corresponded with the prepyloric segment (*Figs. 256, 257*).



FIG. 256.—*Case 8.* Erect position.



FIG. 257.—*Case 8.* Supine position.

OPERATION.—Duodenal ulcer adherent to falciform ligament by a thick band of fibrous adhesions. Rather extensive roughening of pylorus near this. Stomach very much dilated and hypertrophied and mobile. Posterior gastro-enterostomy.

Case 9.—L. S., female. Duodenal ulcer.

Tender point with both right and left lateral shift as compared with supine position. Marked vertical shift (*Fig. 258*).

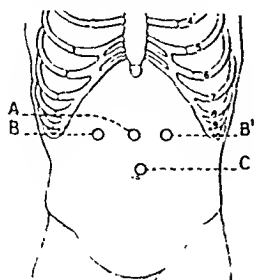


FIG. 258.—*Case 9.* Duodenal ulcer.
A, Supine; B, Right lateral; B', Left lateral; C, Erect.

X RAY.—Erect: with the stomach filled the tender point corresponded with the duodenal cap (*Fig. 259*). Supine: with the stomach filled the tender point again corresponded with the cap, and now lay well to the right of the mid-line (*Fig. 260*). Evidently the filling of the stomach had carried the duodenal cap further to the right.

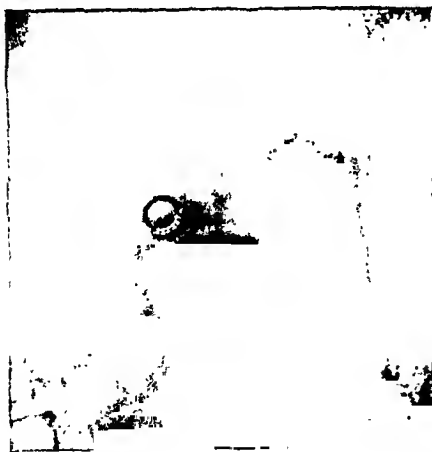


FIG. 259.—*Case 9.* Erect position.



FIG. 260.—*Case 9.* Supine position.

OPERATION.—Duodenal ulcer found immediately under the site of the tender spot, on anterior wall $\frac{1}{2}$ in. beyond the pylorus, with a tuft of organized fibrin over it. Posterior gastro-enterostomy.

Case 10.—A. T., male. Duodenal ulcer.

Tender point with marked vertical shift of 3 in., and slight right lateral shift (*Fig. 261*).

X RAY.—As the stomach filled with barium, the pylorus shifted from a point near the umbilicus, upwards and to the right. The tender point found in the erect position after complete filling was above and to the right of c (*Fig. 261*) and corresponded with the duodenal cap (*Fig. 262*). Supine: the tender point was close to b (*Fig. 261*), and again corresponded with the cap (*Fig. 263*).

OPERATION.—Ulcer on first part of the duodenum, anterior wall, with considerable scarring and stippling, but no adhesions. No gastric ulcer. Posterior gastroenterostomy.

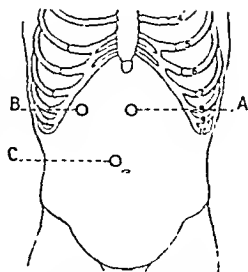


FIG. 261.—*Case 10.* Duodenal ulcer. A, Supine; B, Right lateral; C, Erect.

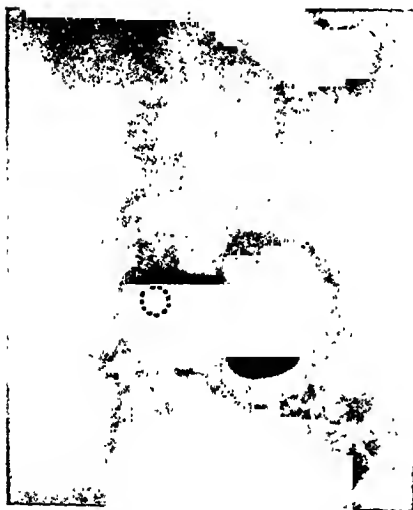


FIG. 262.—*Case 10.* Erect position.



FIG. 263.—*Case 10.* Supine position.

Case 11.—W. E. L., male. Gastric ulcer.

Marked local tenderness. Tenderness in position A (*Fig. 264*), supine, persisted to some extent when the patient was lying on his right side. Also tenderness in position A was still there to a slight extent in the erect posture, though point c (*Fig. 264*) was decidedly more tender.

X RAY.—Erect: after a few mouthfuls of barium deep tenderness at c corresponded with lesser curvature at inner end of a deep persistent incisura. No crater visible. On further filling, the tender point and incisura shifted to the right of the umbilicus. Supine: the tender point was above the umbilicus, and again corresponded with prepyloric position of the lesser curvature.

OPERATION.—Small ulcer on lesser curvature low down. Schoemaker gastrectomy.

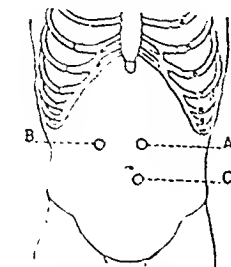


FIG. 264.—*Case 11.* Gastric ulcer. A, Supine; B, Right lateral; C, Erect.

Case 12.—F. B., male. Gastric ulcer.

Tender point in supine position (*Fig. 265A*) disappeared in the right lateral position. (Probably protected by edge of liver.) Marked downward shift in the erect posture (*Fig. 265C*).

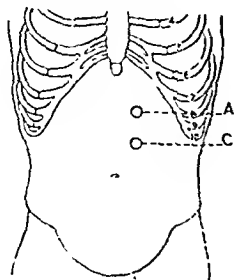


FIG. 265.—Case 12. Prepyloric gastric ulcer. A, Supine; C, Erect. Tender point disappeared in the right lateral position.

X RAY.—Deep tenderness corresponded with a prepyloric ulcer crater, and was found very nearly at points A and C in the supine and erect positions (*Figs. 266, 267*). In one film taken with the patient supine the ulcer and ring did not quite correspond owing to the shift of the stomach with expiration. He was then examined to see whether the tenderness moved with the crater in respiration. This was found to be the case, the ulcer and tender point moving 2 in. between full inspiration and expiration.



FIG. 266.—Case 12. Erect position.



FIG. 267.—Case 12. Supine position.

OPERATION.—Ulcer crater close to the lesser curvature on the posterior wall 2 in. above the pylorus, adherent to the pancreas. On the anterior wall of the stomach at this level, and close to the lesser curvature, was a deposit of organized fibrin. No ulcer in the duodenum. Relation of the ulcer to the liver suggested that our tentative explanation of the loss of the tender point in the right lateral position was correct. The liver margin was lower than usual.

Case 13.—H. B., male. Duodenal ulcer.

Definite point of tenderness in mid-line of epigastrium (Fig. 263). No lateral shift could be demonstrated (on account of very narrow costal angle?). No downward shift in erect posture.

X RAY.—The duodenum was high and did not move downwards with standing or with filling. The tender point corresponded pretty closely with it.

No cutaneous hyperalgesia over the tender point, but the patient states that sometimes when spontaneous pain is bad he cannot bear his shirt to touch the spot.

The skin over the tender area was infiltrated with 1 per cent novocain till it was anæsthetic to pin-prick. There was no obvious effect on the deep tenderness.

OPERATION.—Ulcer on first part of the duodenum, anterior wall. Posterior gastro-enterostomy.

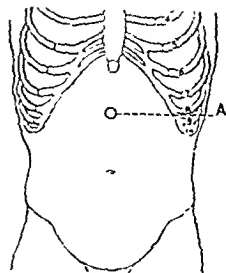


FIG. 263.—Case 13. Duodenal ulcer. A, Supine and erect.

Case 14.—R. C., female. Gastric ulcer.

Tender point in supine position to left of the mid-line. Shift to right in right lateral position; $1\frac{1}{2}$ in. vertical shift in erect position (Fig. 269).

X RAYS.—With the filled stomach, the tender point, which corresponded accurately with the ulcer crater on the lesser curvature, was $\frac{1}{2}$ in. below that found with the stomach empty. Supine: the tender point and crater again coincided, and were in the same position as A (Fig. 269). (Figs. 270, 271.)

OPERATION.—Ulcer on the lesser curvature one-third of the way down from the cardiac orifice. A good deal of fibrous roughening over its anterior surface on the stomach wall. Schoemaker gastrectomy.

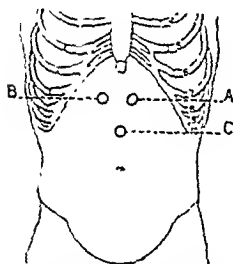


FIG. 269.—Case 14. Gastric ulcer. A, Supine; B, Right lateral; C, Erect.

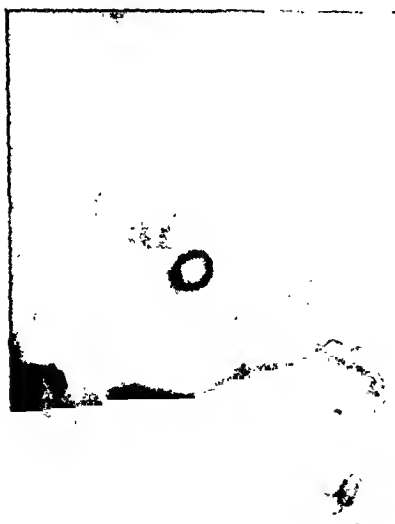


FIG. 270.—Case 14. Erect position.



FIG. 271.—Case 14. Supine position.

Case 15.—T. B., male. Duodenal ulcer.

Well-defined tender point with very slight lateral and vertical shift (*Fig. 272*).

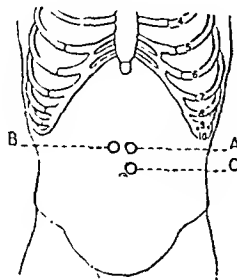


FIG. 272.—*Case 15.* Duodenal ulcer.
A, Supine; B, Right lateral; C, Erect.

X RAY.—Erect: with the filled stomach—the tender point was just to the left of the duodenal cap (*Fig. 273*). Supine: the tender point was slightly higher and again corresponded with the duodenal cap (*Fig. 274*).



FIG. 273.—*Case 15.* Erect position.



FIG. 274.—*Case 15.* Supine position.

OPERATION.—Ulcer on the first part of the duodenum, anterior wall.

Case 16.—E. R., male. Gastric ulcer.

Well-marked tender point. No shift in the right lateral position, but downward shift of $1\frac{1}{2}$ in. when erect (Fig. 275).

X RAY.—Filled stomach: the tenderness corresponded in both positions with those previously found, and with the crater (Fig. 276, 277).

OPERATION.—Large ulcer high up on the lesser curvature extending to the posterior wall. Duodenum normal. Selhoemaker gastrectomy.

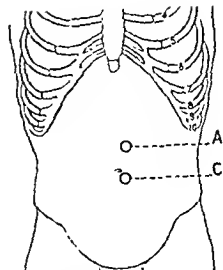


FIG. 275.—Case 16. Gastric ulcer. A, Supine and right lateral; C, Erect.



FIG. 276.—Case 16. Erect position.

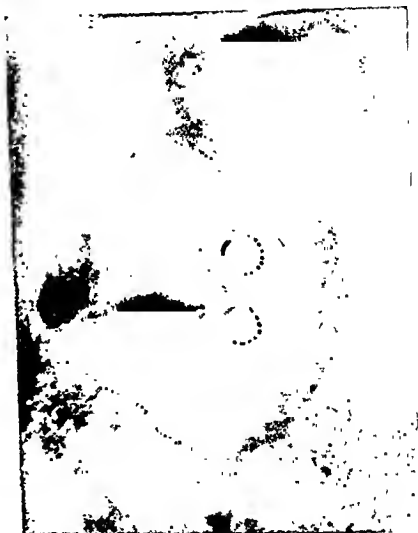


FIG. 277.—Case 16. Supine position.

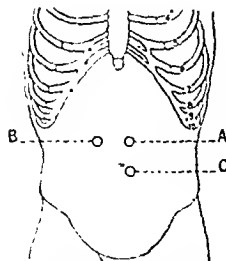


FIG. 278.—Case 17. Gastric ulcer. A, Supine; B, Right lateral; C, Erect.

Case 17.—J. Y., male. Two gastric ulcers.

A single well-defined tender point supine, with definite right lateral and vertical shift of $1\frac{1}{2}$ in. (Fig. 278).

X RAY.—The tender point corresponded with a prepyloric crater in both erect and supine positions. Filling brought it a little lower in the erect posture than that found with the stomach empty.

OPERATION.—One ulcer on the lesser curvature, another on the anterior wall of the stomach $1\frac{1}{2}$ in. above the pylorus.

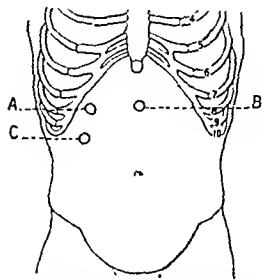


FIG. 279.—Case 18.
Duodenal ulcer. A, Supine;
B, Left lateral; C, Erect.

Case 18.—E. N., female. Duodenal ulcer.

Tender point with a slight right lateral and considerable vertical shift (Fig. 279).

X RAY.—In both erect and supine positions the tender point coincided with the deformed duodenal cap. The shift with altered posture was 2 in.

OPERATION.—Ulcer on first part of the duodenum. Posterior gastro-enterostomy.

Case 19.—E. P., female. Duodenal ulcer.

Tenderness rather more diffuse than usual. Very slight leftward and downward shift with change of posture (Fig. 280).

X RAY.—Erect: the tender point coincided closely with the duodenal cap. Supine: the tender point moved upwards, but rather less than the visualized cap. The duodenal cap appeared to be below the liver edge in both positions (Figs. 281, 282).

OPERATION.—Small duodenal ulcer, anterior wall, first part. Posterior gastro-enterostomy.

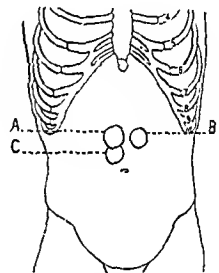


FIG. 280.—Case 19.
Duodenal ulcer. A, Supine;
B, Left lateral; C, Erect.

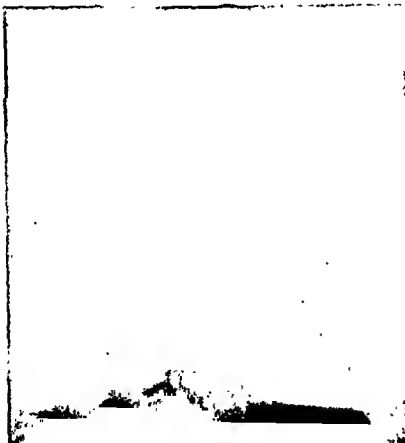


FIG. 281.—Case 19. Erect position.



FIG. 282.—Case 19. Supine position.

DEEP TENDERNESS IN PEPTIC ULCER 391

Case 20.—T. L., male. Duodenal ulcer.

In the supine position tender point to the right of the epigastrium. Marked left lateral and less marked downward shift (*Fig. 283*).

X RAY.—The point of maximum tenderness accurately corresponded with the deformed duodenal cap in both positions.

OPERATION.—Large duodenal ulcer on the first part of the anterior wall, with some adhesions. Posterior gastro-enterostomy.

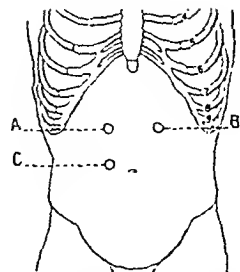


Fig. 283.—*Case 20.* Duodenal ulcer. A, Supine; B, Left lateral; C, Erect.

Case 21.—J. L., male. Gastric ulcer.

Tender point in mid-line of the epigastrium just over a small fatty hernia of the linea alba. (No cutaneous hyperalgesia.) No lateral shift. Slight, but positive, downward shift in the erect position, proving that tenderness was not due to the fatty hernia (*Fig. 284*).

X RAY.—There was about $\frac{3}{4}$ in. vertical shift. The crater was just above the point of deep tenderness in the erect position and a little to the side of it (*Figs. 285, 286*).

OPERATION.—Ulcer on the posterior wall near the lesser curvature half-way up, adherent to the pancreas. Marked thickening and fibrosis of fat on the lesser curvature opposite this, which probably explained why the tender point was to the side of the ulcer. Schoemaker gastrectomy.

Note.—The patient gave no history of pain in the back in spite of adhesions to the posterior parietal peritoneum. The ulcer did not actually invade the pancreas.

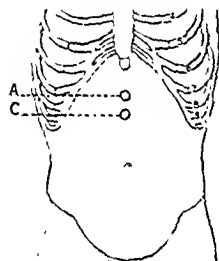


Fig. 284.—*Case 21.* Gastric ulcer. A, Supine; C, Erect. No lateral shift.



Fig. 285.—*Case 21.* Erect position.



Fig. 286.—*Case 21.* Supine position.

Case 22.—J. K., male. Duodenal ulcer.

Usual vertical and right lateral shift of tender point (*Fig. 287*).

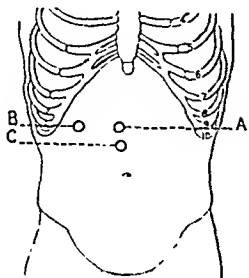


FIG. 287.—*Case 22.* Duodenal ulcer.
A, Supine ; B, Right lateral ; C, Erect.

X RAY.—Erect : the tenderness was over the deformed duodenal cap (*Fig. 288*).
Supine : the tender point was higher, but to the left of the visualized cap, which lay high up under the liver (*Fig. 289*).



FIG. 288.—*Case 22.* Erect position.



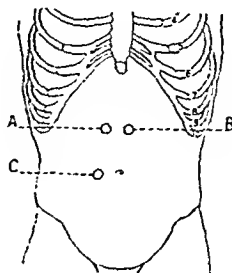
FIG. 289.—*Case 22.* Supine position.

OPERATION.—Marked ulcer on the anterior wall of the duodenum, first part.
Posterior gastro-enterostomy.

Case 23.—M. C., female. Duodenal ulcer.

Tender point with 3 in. vertical shift, but the patient had lost weight, and the abdominal wall and skin were lax. She was also rather confused mentally. In spite of these difficulties, the observations appeared to be sufficiently definite (Fig. 290).

FIG. 290.—Case 23. Duodenal ulcer.
A, Supine; B, Left lateral; C, Erect.



X RAY.—The tender point corresponded with the cap in both positions (Figs. 291, 292).



FIG. 291.—Case 23. Erect position.



FIG. 292.—Case 23. Supine position.

OPERATION.—Ulcer on the first part of the duodenum, anterior wall. Gallstones. Posterior gastro-enterostomy and cholecystectomy.

Case 24.—H. W., male. Duodenal ulcer.

Clinically, definite evidence of shifting of the tender point could not be obtained in the erect posture, because the patient had a trick of tightening up his abdominal muscles and so elevating the stomach on palpation (*Fig. 293*).

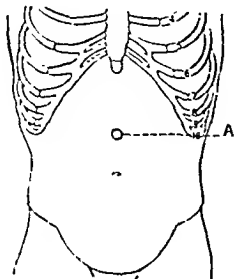


FIG. 293.—Case 24. Duodenal ulcer.
A, Supine and erect.

X RAY.—Erect: with a few mouthfuls of barium the tender spot was at the level of the umbilicus, and was just to the left of the duodenal cap (*Fig. 294*). Supine: there was an upward shift of the tenderness of $1\frac{1}{2}$ in., and the point was again just to the left of the visualized duodenal cap (*Fig. 295*).



FIG. 294.—Case 24. Erect position.



FIG. 295.—Case 24. Supine position.

OPERATION.—Large ulcer on the first part of the duodenum, anterior wall. Contact ulcer on posterior wall. Schoemaker gastrectomy.

DEEP TENDERNESS IN PEPTIC ULCER 395

ANOMALOUS CASES.

Case 25.—C. R., male. Gastric ulcer combined with appendicitis.

Point of maximum tenderness in supine position to the right of the umbilicus. No lateral shift. In the erect position the tenderness shifted down 2 in., but there was still some tenderness in the upper point, and also, rather diffuse, in the area between them (*Fig. 296*).

X RAY.—These points did not correspond with a readily visualized prepyloric ulcer crater on the lesser curvature, but point (2) was just below the third (transverse) part of the duodenum. Pressure over the visualized crater elicited deep tenderness in both standing and supine positions, but these points plainly did not correspond with those shown in the chart (*Figs. 297, 298*).

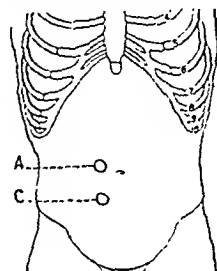


FIG. 296.—*Case 25.* Gastric ulcer and appendicitis. A, Supine; C, Erect.

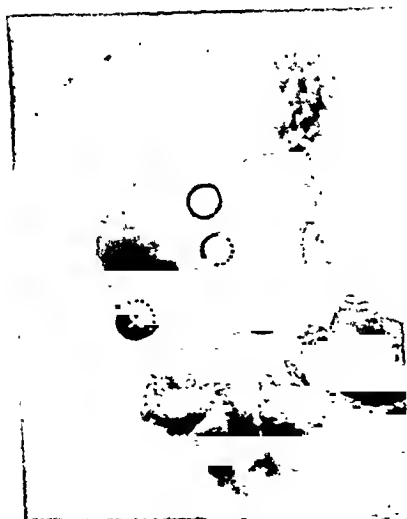


FIG. 297.—*Case 25.* Erect position. X, Point of maximum deep tenderness found clinically.



FIG. 298.—*Case 25.* Supine position. Arrow points to crater of ulcer.

OPERATION.—Large ulcer on the lesser curvature extending to the posterior wall of the stomach. Duodenum normal. A mass of recent adhesions surrounded the appendix, which was not exposed. Schoemaker gastrectomy.

Comment.—Before operation we considered the case to be very anomalous, but the discovery at operation of evidence of a recent attack of appendicitis accounted for the predominant tenderness in an unusually low position. Deep tenderness over the ulcer was present, but was overshadowed by that due to the peri-appendicular peritonitis.

Case 26.—M. A. B., female, age 61. Case with shifting tenderness simulating an organic lesion.

History of pain in the stomach off and on for many years. For the last year or two symptoms have been worse, pain occurring during the night and rousing the patient from sleep. Much flatulence and nausea, but no vomiting. Slight loss of weight and anorexia recently. Pain not related to meals.

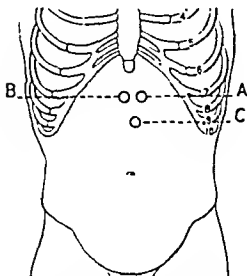


FIG. 299. — *Case 26.* Anomalous case with no organic lesion. A, Supine; B, Right lateral; C, Erect.

ON EXAMINATION.—A well-defined tender point was noted high in the epigastrium to the left of the mid-line (*Fig. 299*), with slight right lateral shift and more marked downward shift in the erect posture.

X RAY.—No ulcer visible, but tender points were marked on the skin, and corresponded with the lesser curvature (*Figs. 300, 301*).

OPERATION.—Stomach, duodenum, and gall-bladder showed no lesion. The appendix was removed, but showed no obvious lesion.



FIG. 300.—*Case 26.* Erect position.



FIG. 301.—*Case 26.* Supine position.

Comment.—This case was one of a small group met with in our investigations in which a typical localized point of shifting deep tenderness was present, without any ulcer or other discoverable organic lesion to account for it. We are unable to explain such cases, but draw attention to them as a caution against the acceptance of a clinically typical point of shifting tenderness as diagnostic of ulcer unless the X-ray appearances confirm the presence of an underlying lesion.

It is as well to point out here that not every visualized ulcer crater is associated with a point of deep tenderness. We have occasionally observed definite ulcer craters, pressure over which elicited no deep tenderness whatever. This absence of tenderness is more common in duodenal than in gastric ulcers, and may be explained by the greater depth of the ulcer from the surface, and the protection afforded to it by the liver.

DEEP TENDERNESS IN PEPTIC ULCER 397

We may also point out that we have not concerned ourselves with superficial cutaneous hyperalgesia in this investigation. We regard it as so inconstant as to be of no value in diagnosis. In Case 13 superficial infiltration of the skin with novocain produced no perceptible diminution of the deep tenderness.

INTERPRETATION OF THE FINDINGS.

We are entirely convinced by these investigations that the correspondence between the tender point and the actual ulcer is real. The coincidence of tender point and ulcer is usually exact, and in most cases in which it cannot be demonstrated on the films as exact, the discrepancy is of a kind which does not exceed the limits of legitimate experimental error. The results obtained on the fluorescent screen were in nearly all cases entirely convincing. We could not in every case reproduce them on films with quite the same precision. Laxity of the skin of the abdominal wall to which the metal rings were attached, varying thicknesses of the abdominal wall, and slight variations in the phase of respiration at the actual moment of taking the film, were all bound to produce slight errors; but the accumulated errors from these causes were in nearly all cases considerably less than the total displacement of both crater and tender point with change of posture. In view of the demonstrable mobility of the first part of the duodenum, notably in spare patients, we were particularly interested, and at first puzzled, to observe what happened when the duodenal cap, deformed by an ulcer, slipped under cover of the liver edge, where it could not be reached directly by the palpating fingers: (1) In some cases the tender point disappeared, particularly when the patient lay on the right side; (2) In others a tender point was found actually to the left of the visualized ulcer now protected by the liver, and usually less easily elicited (*see Fig. 289*). This situation of the tender point to the left of the visualized ulcer, and its diminution in intensity, seemed to us to be explained by partial shielding of the ulcer by the wedge-shaped liver margin from contact with the depressed parietes. These observations differed only in degree from those in cases showing complete disappearance of the tender point, and must have the same explanation.

We are satisfied from these investigations that the correspondence between the point of deep tenderness and the position of the ulcer is a clinical fact which must be accepted and is in most cases readily demonstrable. It should be noted that this is directly in opposition to Mackenzie's observations, which he made before the advent of modern radiological technique.

Our results, therefore, entirely confirm Hurst's recent statements as regards the facts, but our interpretation of them is diametrically opposed to his. Hurst believes that the facts of the relationship between the tender point and the ulcer must force us to the conclusion that the stomach wall where ulcerated is not insensitive, but is endowed with true visceral tenderness. He believes that the seat of this tenderness is the subserous layer of the *visceral* peritoneum over the ulcer. He has thus abandoned a position which, as we have seen, he accepted in his Goulstonian Lectures in 1911.² He has abandoned his earlier belief, not because the body of evidence on which it was based, proving the insensitivity of the exposed viscera to all ordinary painful stimuli, is disproved, but because of these new facts. We hold that there is

nothing irreconcilable between the facts proving the complete insensitivity of the stomach and duodenum, whether ulcerated or not, and the radiological evidence on which Hurst and we ourselves are agreed.

The following case affords direct evidence that the ulcerated stomach is not endowed with visceral tenderness:—

C. H., a very thin man, was admitted to the Manchester Royal Infirmary under the care of one of us (J. M.), with symptoms of pyloric obstruction due to chronic ulcer.

ON EXAMINATION.—The patient had a somewhat diffuse area of well-marked deep tenderness in the centre and right of the epigastrium.

OPERATION (March 22, 1930).—The anterior abdominal wall was infiltrated with 1 per cent novocain. Perfect anaesthesia of the abdominal wall was obtained. A mid-line incision above the umbilicus revealed an extensive fibrinous deposit on the anterior wall of the prepyloric portion of the stomach, corresponding with the area of tenderness, and a large prepyloric ulcer on the lesser curvature adherent to the liver. The inflamed wall of the stomach adjacent to the ulcer was grasped with toothed forceps without any traction being exerted. No pain was complained of. The transverse colon was then drawn up out of the wound, when some pain was felt from the drag on the transverse mesocolon. An avascular space in the transverse mesocolon was selected and a hole torn in it with two pairs of dissecting forceps, care being taken to avoid any further drag on the root of the mesocolon. This procedure caused severe pain, each movement in stretching the mesocolon and widening the hole calling forth a sharp exclamation from the patient. Accordingly a general anaesthetic was administered and posterior gastro-enterostomy completed under ether.

This case proved decisively that the inflamed viscus adjacent to the ulcer was insensitive to direct pressure, though it had been acutely tender when pressed upon through the sensitive parietes. The only possible conclusion is that some part of the abdominal wall is responsible for the deep tenderness. The parietal peritoneum is at once the most sensitive layer and the one in immediate contact with the stimulus. We believe that contact between the parietal peritoneum and the inflamed visceral peritoneum over the ulcer provides the adequate stimulus, though whether that stimulus is mechanical and due to roughening, or chemical and due to bacterial toxins, we can advance no opinion. There is good reason to believe that the tenderness is produced by a process of radiation from the sensitive parietal peritoneum to the skin and subcutaneous tissue over it supplied by the same cerebrospinal nerves. The belief that the tenderness is not localized by the sensorium in the parietal peritoneum, but in the more superficial structures, is based on the earlier studies of one of us (J. M.) on shoulder-tip pain.^{5,6}

Our more immediate concern, however, is to establish the parietal, as against the visceral, peritoneum as the point of origin of the impulse, and of the cerebrospinal, as against the splanchnic, nerves as its afferent pathway. On these points it appears to us that Hurst has expressed views which lead him to an untenable position. He claims that the only possible interpretation of his radiological findings is a true visceral tenderness. According to his view, the same afferent splanchnic nerve-endings in the region of the ulcer must be stimulated whenever pressure is applied over the ulcer. He therefore claims by implication for the splanchnic nerves an accuracy of spatial localization transcending anything that we know of elsewhere in the

body. For even in somatic sensory nerves accurate discrimination between the different points to which a stimulus may be applied depends upon the stimulation of different sets of nerve-fibres.

How the afferent splanchnic nerves can be supposed to appreciate slight changes in the relationship between an ulcer and the abdominal wall, we are quite unable to see. Yet this power of localization, as we have demonstrated is beyond dispute. It must of necessity involve the stimulation of fresh nerve-endings in each position of the ulcer. These nerve-endings can only be in the parietal peritoneum.

SUMMARY AND CONCLUSIONS.

1. The theories hitherto accepted as to the mechanism of deep tenderness associated with gastric and duodenal ulcers are discussed.

2. An investigation of 25 patients with chronic ulcer associated with localized deep tenderness showed that the area of deep tenderness on the anterior abdominal wall corresponded generally with the ulcer crater, and shifted its position with the ulcer.

3. These observations are quite incompatible with Mackenzie's theory of a viscerosensory reflex.

4. A case is recorded proving that the stomach, when ulcerated and exposed to direct pressure by an incision under local anaesthesia, is insensitive to pressure.

5. Deep tenderness is explained as due to stimulation of the sensitive parietal peritoneum by contact with the inflamed area of stomach or duodenum at the site of the ulcer, and radiation from the nerves of the parietal peritoneum to the more superficial branches of the same sensory cerebro-spinal nerves.

REFERENCES.

- ¹ MACKENZIE, *Symptoms and their Interpretation*, 4th ed., 148.
- ² HURST, *Goulstonian Lectures on the Sensibility of the Alimentary Canal*, 1911, 65.
- ³ HURST and STEWART, *Gastric and Duodenal Ulcer*, 1929, 163.
- ⁴ KINSELLA, V. J., *Med. Jour. of Australia*, 1928, Jan. 21.
- ⁵ MORLEY, J., "The Clinical Significance of Shoulder-tip Pain", *Clinical Jour.*, 1925, liv, 617.
- ⁶ MORLEY, J., "The Significance of the Afferent Impulses from the Skin in the Mechanism of Abdominal Pain", *Lancet*, 1929, ii, 1240.

ACUTE OSTEOMYELITIS OF THE SPINE.

BY THE LATE H. W. CARSON,

SOMETIME SENIOR SURGEON AT THE PRINCE OF WALES'S GENERAL HOSPITAL, LONDON.

THE occurrence of acute osteomyelitis of the spinal vertebrae following upon some slight lesion is not common, but the difficulty of diagnosis, the severity of the symptoms, and the prolonged convalescence make this disease one of great importance, and form my reason for publishing in some detail the four cases described below. My interest in the condition was aroused by the case of my friend Dr. C. (*Case 1*), and subsequently I have collected details of three others, two of which occurred in the practice of Mr. J. Everidge and the other in that of Mr. G. W. Thomas, of Wakefield. I am much indebted to these two surgeons for the help they have given me, and I am particularly grateful to Dr. C. P. Symonds for his analysis from the neurological aspect of *Case 1* and for the assistance he so ungrudgingly gave through this very difficult and prolonged illness.

These four cases show certain similarities, and may well be classed in one group. The initial cause in three was the same—a boil—and in the fourth the boy had a subcutaneous abscess in the coccygeal region containing pus, giving a pure culture of the *Staphylococcus aureus*; it is possible that this was a secondary abscess, the original focus not being apparent. A feature common to all was an infection of the urinary tract. One patient lost his kidney from multiple abscess. The boy had his kidney explored, though the reason for suspecting it is not mentioned in the notes, and in the other two cases prostatitis occurred, one going on to abscess.

The invasion of the vertebral bodies took place at very varying intervals after the primary lesion. *Case 3* had no spinal symptoms till nine months after the lesion was confirmed by X rays, ten months after the beginning of her illness. *Case 1* had symptoms four weeks, and the lesion was confirmed by X rays four months from the beginning. The times for *Case 2* are three months and fourteen months, respectively, several earlier X-ray photographs proving negative; *Case 4* started with lumbar pain, and the X ray showed a lesion two months later, a radiogram taken seven weeks earlier proving negative. An abscess formed in three cases, in *Case 2* not till four years after the initial lesion. In all the vertebral body was affected, the lumbar in three cases and the cervical in one. It may be said, therefore, that these four cases, in spite of the severity of the onset, were really examples of subacute osteomyelitis, and contrast very definitely with, for instance, a series of 24 cases collected by Makins and Abbott,¹ of which 8 were under their own care and all died, the mortality in the 24 cases amounting to 70 per cent.

The disorder seems to have been studied mostly in France at first, and the list of French authors is a long one, starting from the classic paper by Lannelongue.² He had seen only one case of osteomyelitis of the lumbar vertebra. Since then, many cases have been reported and several varieties

are described. Labyerie³ recognized four varieties: (1) A very acute form, which is a true osteomyelitis; (2) A subacute form, limited in extent, which ends in an abscess; (3) An acute or subacute form, which is rather osteoperiostitis and ends in resorption; (4) A chronic form, which is an ankylosing osteo-arthritis. He considered that the true cause was the introduction of a 'pathological agent' into the general circulation. He refers to boils and minor infections round the finger-nails as a cause, and says the micro-organism is the *Staphylococcus aureus*.

Mathieu⁴ considers the acute and subacute forms the most important. He says that the acute form is generally due to a common pyogenic microbe, of which the most frequent is the *Sta. aureus*, while the subacute form generally complicates an infectious disease such as typhoid. He also recognizes three other types: (1) Phlegmonous periostitis; (2) External periostitis giving rise to an abscess between the periosteum and the muscles; and (3) Epiphysial or juxta-epiphysial osteomyelitis. He says that injury causes 25 per cent of the cases, and places a skin lesion, such as a boil, in a secondary position; in this he follows Lannelongue.

Etiology.—The surgeons of the eighteenth century were not greatly concerned with the cause of disease, and in Pereivall Pott's account (1779) of the disease of the spine which was eventually called after him, there is no attempt to explain its origin. The tuberculous nature of Pott's disease was suspected by the French surgeons in 1825, and gradually the name 'Pott's disease' became synonymous with tuberculous disease of the spine. Thus tubercle as a cause of acute or subacute disease of the vertebra overshadowed any other possible cause, and in my student days we were taught that there was a 'senile' tuberculosis occurring in late adult life. No real evidence was ever produced that these cases were actually tuberculous, and the question may be asked whether they did not include among them cases of infective osteomyelitis, while the more daring may question the occurrence of senile tuberculosis of the spine altogether.

Many cases have now been recorded of acute osteomyelitis of the vertebra following certain specific infections—for example, typhoid fever, pneumonia, gonorrhœa, and rarely syphilis. In the four cases under review, all due to a staphylococcal blood infection, the determination of the lesion to the bodies of the vertebra is very difficult to explain. Many authors have suggested injury as the exciting cause, and cases have been recorded where there has been a clear history of recent local injury (Fraser and McPherson,⁵ J. Ramsay Hunt,⁶ Lannelongue,² and others); but an injury to the body of a vertebra must be rare and there is no suggestion of an injury in the four cases I report. Infection is due to the *Sta. aureus* in the opinion of all observers, and I have been fortunate in obtaining evidence of a pure culture of the staphylococcus in the blood in all my cases.

Site of Disease.—The bodies or the laminae of the vertebrae may be affected. If the body is the seat of the disease, it seems to attack those of the lower half of the spine. Mathieu⁴ puts the order of frequency as lumbar (53 per cent), then dorsal, cervical, and sacral, and says that the posterior arch is more often affected than the body except in the neck. Labyerie³ says that there is a remarkable predilection for the lumbar region, and Hahn⁷

reported 41 cases in which the cervical region was affected 7 times, the dorsal 12, the lumbar 17, and the os sacrum 5.

Symptomatology.—The symptoms vary according as the body or lamina is affected, and there are local variations due to the part of the spine concerned. The local manifestations may be obscured by the severity of the general symptoms. The onset may be very severe—rigors, high fever, great prostration, with delirium, sleeplessness, and diarrhœa. A high leucocytosis is the rule. Attention may be drawn to the spine by complaints of agonizing lancinating pains radiating along the affected nerves, or by the rigidity of the muscles guarding the infected area, but there may be no local signs of inflammation unless the laminae are affected, when diagnostic complications may result by the supervention of signs of spinal meningitis. In very acute cases a fatal toxæmia occurs and death ensues in ten to twenty days from the onset. In less acute cases, among which those here recorded should be included, implication of the spine, or at least direct evidence of it, may be delayed for very variable periods.

The time after the onset of the illness at which backache was complained of was 19 days (*Case 4*), 4 weeks (*Case 1*), 3 months (*Case 2*), and 9 months (*Case 3*); and confirmation by the Röntgen rays was possible in 2 months, 4 months, 14 months, and 10 months, respectively. An abscess occurred in three of the four cases: at 4 months (*Case 1*), 10 months (*Case 3*), and 48 months (*Case 2*) after the onset. It is curious that all cases had an infection of the urinary tract, in *Case 3* severe enough to lead to nephrectomy. In the early stages it seems that one must rely upon two main symptoms, rigidity of the muscles locally and radiating pains, and that these latter symptoms are not easy to assess, is seen by Dr. Symonds' notes in *Case 1*. In the early stages, as is shown by *Cases 2* and *4*, skiagrams are not helpful; they must be taken repeatedly.

Differential Diagnosis.—The important differentiation is from tuberculous disease of the spine, and this is not difficult as a rule. Infective osteomyelitis of the spine affects adults more often than children, is preceded by some disorder such as a boil (though it must be remembered that a trifling ailment like a boil may be overlooked or forgotten), pneumonia, or typhoid fever, and has a severe onset with high temperature and extreme prostration. The three main symptoms of tuberculous disease are deformity, cold abscess, and paralysis, of which deformity is usually the first sign. In infective osteomyelitis deformity is rare owing to the buttressing of the weak spot by new bone ('bees de parroquet'). This is well seen in the radiograms. Pain, and especially lancinating pain, is characteristic of infective osteomyelitis, and is not relieved by rest as in Pott's disease. Abscess is more frequent in infective osteomyelitis than in Pott's disease, but the spinal cord is not affected except in disease of the posterior arch.

Prognosis.—In the acute variety mortality is very heavy. As mentioned above, Makins and Abbott¹ give a mortality of 70 per cent. Mathieu⁴ reports Gridel as giving a mortality of 46 per cent, and he himself considered osteomyelitis of the body of the vertebrae as fatal. One finds in the literature reports of many recoveries, and it is probably necessary to draw a definite distinction between acute and subacute conditions as far as prognosis is concerned, the latter usually running a long course ending in recovery.

CASE REPORTS.

Case 1.—Dr. R. C. (Under the care of Drs. Carpmacel and Stewart. Dr. Symonds and Mr. Carson in consultation.)

July 31, 1929.—Two carbuncles opened on left side of neck. They did not seem of importance and soon healed, and the patient left for a holiday at Bexhill, and I (H. W. C.) left also.

Aug. 14.—Dr. Symonds reported: "Felt suddenly seedy. Temperature 103°, headaches, vomiting, and generalized pains."

Aug. 23.—Dr. Symonds reported: "Fever has continued about 103°, with an evening rise and profuse sweats. For the last five or six days the leading symptom has been intense pain in the back of the neck; this is diffuse, spreading down to the right shoulder and up to the head. Swallowing is difficult on account of pain. Three or four days ago a profuse crop of herpes labialis."

ON EXAMINATION.—The patient was gravely ill and exhausted, but mentally clear, with some physical reserve. Passive movement of head in all directions, free and painless within a very small range. No definite point of tenderness in any cervical spine, but tenderness in the transverse processes beneath the sternomastoid about the 3rd cervical. No tenderness or œdema on posterior wall of pharynx. No glands: nothing in chest, heart, or abdomen. All tendon-jerks present and equal. No defect in the cranial nerves, except that on phonation there was no movement of pharynx. White blood-count, 17,000; urine, trace of albumin; blood culture being done.

DIAGNOSIS.—Staphylococcal septicæmia. ? Arthritis. ? Osteomyelitis in neck—probably the former.

Aug. 27.—Dr. Creed reported: "White blood-count, 17,000; polymorphs, 84 per cent. Urine, a trace of albumin, a few colonies of *Sta. aureus*. Blood culture, *Sta. aureus* in pure culture after forty-one hours in each of eight tubes inoculated."

Sept. 5.—On returning from my holiday, I (H. W. C.) found an urgent message. Acute retention of urine had occurred during the last forty-eight hours. I found some induration in the left neck, which seemed to be subsiding, and a curious alteration in the voice, though nothing was seen on examination of the pharynx. The prostate was acutely tender.

Dr. Symonds saw him, and reported: "After he was last seen the patient had much pain in the left shoulder and burning pain and tingling down the ulnar side of the arm, subsequently paralysis of muscles around the shoulder and a large mass of glands beneath the sternomastoid just above the clavicle. During the past forty-eight hours, retention of urine with much spasm and pain. Has had to be catheterized. Prostate very tender (Carson). Fever still high. General condition pretty fair."

EXAMINATION.—The left pupil was smaller than the right, otherwise the cranial nerves were normal. There was weakness and wasting of the left deltoid and spinati, otherwise no motor defect. The supinator- and biceps-jerks were absent on the left and present on the right; the triceps-, knee-, and ankle-jerks were present and equal: the abdominals were all four present and equal; and the plantar responses were flexor. The tongue was moist, the general condition fair, and nothing abnormal was found in the chest or abdomen.

Sept. 6.—Suprapubic drain. Pure staphylococci again found in blood and urine. The bladder was drained for a fortnight, at the end of which time the prostatitis had subsided.

Oct. 9.—Dr. Symonds reported: "Since last seen, pain and weakness in the left shoulder improved, but recurrence of pain developed in the neck, travelling down the right arm with paralysis of right deltoid. Had a suprapubic drainage. Carson thought he has had a prostatic abscess. Subsequently improved, but still has a little irregular temperature and occasional bouts of severe pain in neck and arms. Blood culture now sterile."

Nov. 12.—Dr. Symonds reported: "Temperature running high; blood culture negative; blood-count shows secondary anæmia; white cells, 10,000. Looks

better and stronger, but fallow. Has occasional pain behind the right scapula to about the insertion of the right deltoid, but less severe than before. Weakness and wasting of the right deltoid and biceps; absent right biceps-jerk. I could detect no other physical signs. To consult Ryle about possible value of blood-stream disinfectant."

Nov. 19.—Dr. Symonds reported: "Temperature still up. Tender point in right scapula still present, quite definite. Tender over transverse process of 5th cervical vertebra both sides. Heart, ? soft, to-and-from murmur aortic area."

Nov. 28.—Seen in consultation with Drs. Symonds, Carmichael, and Stewart. Some discomfort in swallowing. Swelling noticed in posterior pharyngeal wall. X-ray photograph (Dr. Worth) shows necrosis and considerable destruction of the 4th cervical vertebra (Fig. 302). Decided to try immobilization of cervical spine.

Dec. 1.—Immobilization by plaster jacket.

Dec. 6.—The pharyngeal swelling is now more right-sided, and is large enough to hide the arytenoids. No swelling of neck or deformity or fixation of cervical spine.

OPERATION (Dec. 14).—Operation at St. Peter's Nursing Home, Leigham Court Road. Dr. Frankis Evans gave anaesthetic; Messrs. W. E. Tanner, Carmichael, and Stewart assisted. A quantity of thick pus was liberated from beneath the anterior common ligament by incision behind the sternomastoid. Pure *St. aureus* on culture.

SUBSEQUENT PROGRESS.—The temperature fell to normal very soon, but the dressings were most painful owing to some irritation of the posterior auricular nerve which may have been exposed in the wound. The patient improved very rapidly, sleeping well and putting on weight.

May 1, 1930.—Seen. The sinus has completely closed. There is occasional pain under the right pectoral muscle on certain movements, and a tender



FIG. 302.—Case 1. Showing condition of the 4th cervical vertebra.

spot on top of the right shoulder. Flexion of neck is good, rotation less good. Limitation of movement of right shoulder chiefly due to loss of power in deltoid, but abduction causes radiating pain down the arm. There is a very little lateral deviation to left of cervico-dorsal spine. Is wearing a leather jacket, and is back at work.

At no time has any sequestrum appeared, nor is there any evidence of such in the latest X-ray picture. There is a well-marked spur of new bone ('bee de perroquet').

June 24.—Felt as though some small body was stuck in the back of his throat, felt with his finger, started coughing, and brought up two minute sequestra.

May 13.—Dr. Symonds wrote: "If I might add a note upon the points of medical interest, the situation on Nov. 19 was to my mind that the continued pyrexia could only be explained satisfactorily by supposing one of two conditions to exist—either an infective endocarditis or a hidden suppurative focus. Against infective endocarditis was the absence of any definite bruit."

"It was this which determined me, after I had had a talk with Ryle about him, to have him X-rayed round about the parts where he was still complaining of pain."

Looking back, I feel that my attention at this stage should have been drawn again to the possibility of a central lesion to explain the bilateral involvement of 5th and 6th cervical roots, rather than two separate paravertebral lesions.

"Earlier on I had suspected an osteomyelitis of the vertebræ, but had not been able to conceive such a condition producing bilateral root involvement without at the same time compressing the spinal cord, and of this there was no clinical evidence at any time, once the retention of urine had been explained by you upon a local basis.

In retrospect there is another point to which Ryle first drew my attention in the history of the case, which I think is of considerable clinical importance, and that is the appearance of those enlarged glands on the left side of the neck in the early stages of the illness. Ryle's comment upon this was that one would not expect enlargement of the glands in a case of staphylococcal septicæmia, except by direct spread from a suppurative focus along the lymphatic channels: hence this enlargement, though transitory, pointed to some latent suppurative focus in the neck.

"I might add that at the time of my examination on Nov. 12, although I had come to the conclusion that we were probably dealing with a collection of pus somewhere in the bones of neck or shoulders, I was quite unprepared for the picture revealed by X rays, seeing that active and passive movements of the neck were relatively free and painless and there was no tenderness to pressure upon any of the cervical spines. The tenderness to pressure upon the mid-cervical transverse processes, however, which I first noted in my first examination, was a constant feature throughout."



FIG. 303.—Case 2. X-ray appearance of 2nd and 3rd lumbar vertebrae.

Case 2.—Dr. S. (Under the care of Mr. Everidge.)

HISTORY.—At the end of 1921, while on holiday convalescing from boils in the neck, the patient was seized with acute retention of urine. He returned to London by ambulance, and was seen by Mr. Everidge, who found he had a prostatic abscess, for which he operated by the perineal route within an hour or two of his arrival in London. Recovery was rapid, but two or three weeks later he developed an acute mastoid, for which Mr. Jenkins operated. He went down to Torquay to convalesce, but while there developed severe pain in the left flank, which became so acute that he had to return to London. No cause for this pain was discovered, nor did the X rays repeatedly taken show anything abnormal. During the succeeding months his pain became worse, and wasting occurred of the left thigh muscles. Finally, X-ray examination showed partial destruction of the bodies of the 2nd and 3rd lumbar vertebrae (Figs. 303, 304). He was fitted with a spinal jacket, and his subsequent history is as follows:—

May, 1923.—Superficial abscess over the mastoid region. Healed in ten days.

End of May to middle of August, 1923.—Plaster-of-Paris jacket.

September, 1924.—Herpes zoster.

June, 1925.—Abscess in the right lumbar region extending down into buttock. Operation. Sinus healed by October, re-opened in December, and finally healed in March, 1926. (Ultra-violet rays.)

April, 1926.—Abscess of vertex of the skull after knocking his head on a beam. Large sequestrum removed, exposing the dura.

May, 1927.—Abscess of the right frontal bone followed injury. Sequestrum removed in July.

April-May, 1929.—Pain and swelling in the lumbar renal region. In bed a month. Condition subsided without operation.

Since July, 1929, he has been continuously at work, and in good health apart from backache and some stiffness in the lumbar region. He ascribes his freedom from abscesses since July, 1927, to a course of intravenous injections of perchloride of mercury.



FIG. 304.—Case 2. Same as Fig. 303: lateral view.

Case 3.—Mrs. B., age 43. (Under the care of Mr. G. W. Thomas.)

Nov. 13, 1927.—What appeared to be simply a small 'boil' on the chin (halfway between the lip and the lower edge of the jaw) was pricked with a sterile needle. No pus found.

Nov. 15.—Redness and infiltration round the 'boil'. Free crucial incision. Fomentations.

Nov. 16.—Extension of swelling, etc., to whole of the chin and lower lip. Free incisions in chin and lip. Condition evidently 'carbuncle' of the chin.

Nov. 18.—Extension to the left side of the mouth and below chin. Further incisions (left side of the chin and just below jaw in the mid-line). Collosol man-

ganese 15 c.c. Antistaphylococcus serum 10 c.c. Wounds treated with H_2O_2 , collosol, and flavine.

Nov. 19.—Serum 10 c.c.

Nov. 20.—Collosol manganese 1 c.c.

Nov. 21.—Impaired resonance and weak breath-sounds in the lower half of the right chest posteriorly.

Nov. 22, 24, and 27.—Collosol manganese 1 c.c.

Dec. 1.—Right base explored: 5 oz. of clear fluid (this was not examined). Collosol manganese 1 c.c. Patient still very ill.

Dec. 4.—Collosol argentum 4 c.c. intravenously.

Dec. 5.—Collosol manganese 1 c.c.

Dec. 7.—Collosol argentum 5 c.c. intravenously.

Dec. 11.—Collosol argentum 6 c.c. intravenously.

The patient began to improve about this time. Wounds all healed towards end of December.

Jan., 1928.—Condition not entirely satisfactory, but no definite symptoms.

Jan. 18.—Blood examination showed a marked polymorphonuclear leucocytosis.

Jan. 19.—Right chest explored—negative.

Towards the end of the month some pain and tenderness were noted in the right side of the abdomen.

Feb. 3.—Right kidney enlarged and tender. Urine: Much pus, pure culture *Sta. aureus*, no T.B.

Feb. 20.—Right nephrectomy (lumbar)—some perirenal suppuration. Large tube drain.

May 21.—Urine: Few pus cells, few *B. coli*, profuse culture *Sta. aureus*. (Also on March 30 and April 24.)

June.—Sinus following removal of tube healed about this time.

Sometime after the nephrectomy (about April), the left kidney was a little enlarged and tender, but very fortunately settled down.

July 24.—Urine: Very few pus cells and cocci. The pathologist's report on the kidney said there were numerous pyæmic abscesses in section, and from the pus of one of them a pure culture of *Sta. aureus* was grown.

The patient now seemed well and went away for a holiday, during which she developed backache (lumbar). This gradually became severe and she required veramon tablets, etc., for sleep.

September.—In the middle of this month the nephrectomy wound re-opened and began discharging pus. Examination of the latter showed staphylococci, and culture gave a pure growth of *Sta. aureus*. Guinea-pig inoculated, but when killed showed no signs of T.B. Abdominal and pelvic examination was negative, and apart from the backache there was nothing suggesting spinal trouble. However, as the sinus persisted, an X-ray examination was made and showed "a very distinct lesion of the 2nd lumbar vertebra, which could quite well be an infective osteomyelitis." So I injected the sinus with lipiodol, and this was shown by X rays to pass down a track leading to the 2nd lumbar vertebra and a little way over to the left side. A large probe was also found to pass just to the same spot, the sinus being about 9 in. long. This made the diagnosis quite complete.

December.—The patient was fitted with a Thomas's back support, and allowed to get about, the pain having subsided.

Jan. 30, 1929.—X ray (Fig. 305). Traces of lipiodol still present—a little more erosion of the vertebra.

March 3.—A small sequestrum came away, after which the discharge diminished and the sinus was quite healed on March 14. The patient continued to wear the support for some time, and has remained quite well since—free from all symptoms.

June 4.—X ray. "Condition appears to be much improved—no real callus formed, however. Trace of lipiodol still present."



FIG. 305.—Case 3. Showing erosion of 2nd lumbar vertebra.

A series of skiagrams of the whole skeleton showed it to be perfectly normal, with the exception of the left lower limb, which gave the remarkable appearance seen in *Fig. 307*. The femur is noticeably shortened and is grossly deformed in contour. The neck is at a right angle to the shaft, and below the neck large irregular bony protuberances are seen to the inner side of the shaft. The normal distinction between cortex and medulla is almost entirely lost, and especially towards the upper end of the bone the appearance is that of numerous, irregular, translucent areas separated by narrow septa of denser bone.



FIG. 307.—X-ray appearance of femur and tibia.

The tibia shows a uniform, fusiform enlargement, greatest at the mid-point of its length. The head and adjacent parts are very translucent; the rest of the bone shows no resemblance to normal structure and has been replaced by an intricate filigree or basket-work of bony trabeculae outlining translucent spaces. It is noteworthy that the fibula is entirely free from the disease.

OPERATION.—Disarticulation through the hip-joint was done, and recovery was without incident.

PATHOLOGICAL FINDINGS.—The bones were prepared by dissection immediately after amputation. No affection of the soft parts was found, but it was evident that the periosteum was greatly thickened. A section of each bone was made, and the appearance is very accurately depicted in *Figs. 308, 309*. The altered contours of the bones are well seen in section: the cut surfaces are composed, for the most part, of a white fibrous tissue, which was very tough and gritty to the knife. The cut surfaces are marked here and there by small trabeculae of bone and by points of hæmorrhage, and are flecked by spots of hyaline cartilage, which is seen in considerable masses in the upper third of the tibia. The only cyst disclosed by section of the bones is a small cavity at the mid-point of the femoral shaft, lined by a tough fibrous membrane.

A large number of blocks of the tissue were taken, and for these I am indebted to the Pathological Laboratory of the University of Manchester. Out of many examined, those have been selected

for illustration which appeared to show the essential features of the disease in this case (*Figs. 310–317*). Evidence of absorption of bone is to be found in most of the microphotographs. It is very difficult to be sure that any of the bone is the original structure, and it is highly improbable that in a

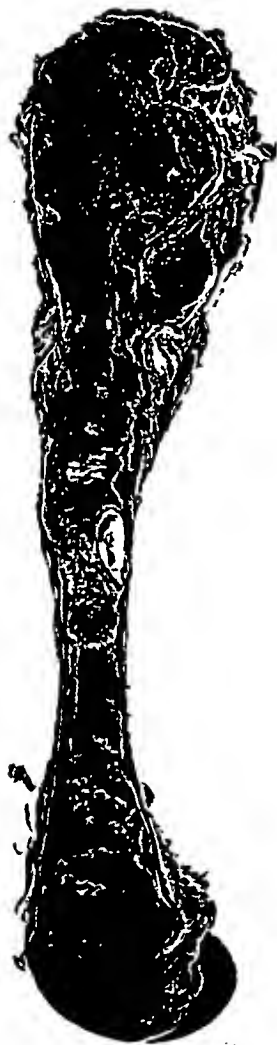


FIG. 308.—Vertical section of the femur. ($\times \frac{1}{2}$.)



FIG. 309.—Vertical section of the tibia. ($\times \frac{1}{2}$.)

specimen so old and so advanced any portion of the original bone remains. It is never easy to determine the old original bone, and the difficulty is due to the fact that newly-formed bone may, in its turn, be broken down and removed. Dawson,² writing of this difficulty, says that he regards as characters of old bone: (1) Haversian systems with definite concentric and intervening parallel



FIG. 310.—The stage of destruction, showing bone being eaten away by osteoclasts. ($\times 75$.)



FIG. 311.—A further stage in the destruction. Large osteoclasts surround the almost disintegrated fragments. The early loose and very cellular tissue is shown. ($\times 150$.)

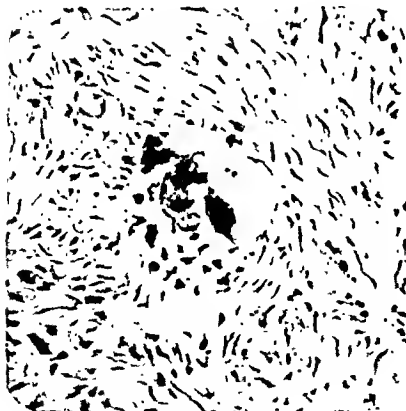


FIG. 312.—A later stage of *Fig. 311*, showing a somewhat denser fibrous tissue. ($\times 150$.)



FIG. 313.—The final result of the destruction. Many large syncytial masses lying in a highly cellular fibrous stroma. ($\times 75$.)

lamination; (2) Bone cells uniformly distributed and spindle-shaped, with definite, regular, almost parallel canalicular processes from their sides; (3) Evidences of calcification. On the other hand, he accepted as newly-formed bone that showing: (1) A homogeneous appearance or only slight peripheral

lamination; (2) Cells closely arranged axially and with irregular and radiating processes; (3) Transition at its fibrillated ends into fibrous tissue; and (4) A minimal calcification in the axial portion.

The portion shown in *Fig. 310* is taken from just beneath the thickened periosteum, and the bone undergoing destruction may possibly be old bone.

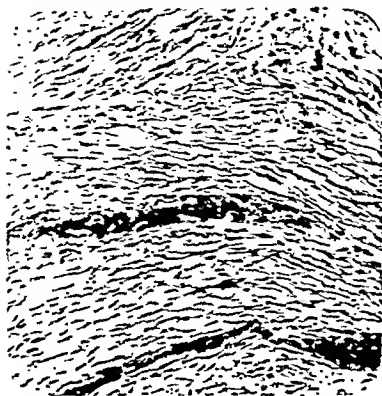


FIG. 314.—Dense, well-formed fibrous tissue showing the beginnings of trabeculae of new bone. ($\times 75$.)



FIG. 315.—A later stage in the formation of new bone. Many small, mononucleated osteoblasts applied to the growing edge. ($\times 75$.)



FIG. 316.—A portion of the hyaline cartilage. ($\times 110$.)

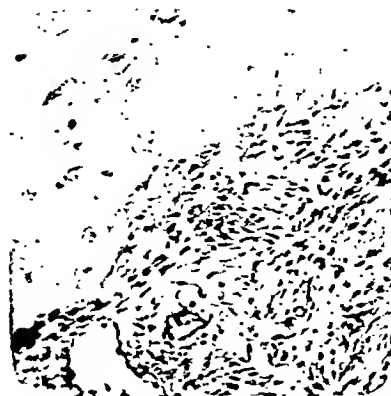


FIG. 317.—The growing edge of the cartilage, showing its origin from the fibrous tissue. ($\times 110$.)

The transition from bone to fibrous tissue is shown in *Figs. 310-312*, until in *Fig. 313* we reach the condition of a loose fibrous tissue enclosing numbers of large multinucleated giant cells. This tissue, under a high power, suggests great activity of growth, and mitotic figures are common. The resemblance

to an actively growing spindle-celled sarcoma is very close. The ultimate fate of this young connective tissue is the formation of very dense, well-formed fibrous tissue of which the bulk of the altered bone is formed. In many places the formation of trabeculae of new bone is seen; *Fig. 314* shows three such areas in which the ground-work of new bone is being laid down in striae enclosing the future bone cells in its substance. *Fig. 315* shows a further stage of this process, and it will be noted that the new bone has not the orderly, laminated appearance of original bone.

An unusual feature of the case is the formation of masses of hyaline cartilage. This, as seen in *Fig. 316*, is a true hyaline cartilage, but is crude and imperfect in structure. It is formed by direct growth out of fibrous tissue, and this development is shown in *Fig. 317*. On the right-hand side of the section an area in which chondromucin is being laid down in parallel striae is well shown. There is nowhere any indication that the hyaline cartilage represents a stage in the formation of new bone.

It is seldom that the opportunity is given to investigate fully an osteitis fibrosa of at least twenty years' standing, and this specimen appears to illustrate all stages of this weird disease. The chief impression left by a study of the sections is that of an intense cellular activity very closely resembling that of sarcoma. It would appear, however, that in osteitis fibrosa this activity is directed to the formation of a dense fibrous tissue and very seldom oversteps the boundary into 'new growth'. In this we have an obvious contrast between osteitis fibrosa and the closely related osteitis deformans, since in Paget's disease the eventual transition into sarcoma is a common terminal event.

REFERENCES.

- ¹ KNAGGS, "Osteitis Fibrosa". *Brit. Jour. Surg.*, 1923, x, 487.
- ² DAWSON and STRUTHERS, "Generalized Osteitis Fibrosa", *Edin. Med. Jour.*, 1923, Oct.
- ³ BLOODGOOD, "Bone Cysts". *Ann. of Surg.*, 1910, lii, 148.
- ⁴ ELSLIE, "Fibrocystic Disease of Bone", *Brit. Jour. Surg.*, 1914, ii, 32.

A CASE OF PRIMARY ADAMANTINOMA OF THE TIBIA.

By ALFREDA H. BAKER,

CONSULTING SURGEON TO HOUNSLOW HOSPITAL;

AND L. M. HAWKSLEY,

ASSISTANT PATHOLOGIST TO THE CANCER HOSPITAL (FREE), LONDON.

CLINICAL NOTES.

(A. H. BAKER)

A MAN, age 46, a locomotive driver, was first seen on Sept. 15, 1930, in the Out-patient Department of Hounslow Hospital. He was complaining of pain in the lower part of the left leg.

HISTORY.—The patient gave a history of having been struck on the left shin by a bar of iron ten months previously. This caused only temporary inconvenience, and there was no wound of the skin. Three months before I saw him he had received another blow on the same place, also from a bar of iron. This injury was very painful, but, again, there was no cutaneous injury. The severe pain lasted for about two hours, and left a dull gnawing pain for a fortnight. Six weeks before he came to hospital he stumbled while carrying a heavy weight on his back, and the jar to his leg caused the pain to return. Local applications of embrocation gave no relief, but he continued working until ten days before his attendance at hospital. He was then seen for the first time by Dr. V. C. Montgomery, who sent him to hospital for an X-ray examination of the leg.

ON EXAMINATION.—A slight swelling of indefinite outline over the lower third of the left leg on its inner and anterior surfaces was observed. The skin appeared to be quite normal; there was no mark of any wound, recent or healed. The swollen area was slightly tender and warmer to the touch than the surrounding skin. Pressure on the sole of the foot caused pain in the leg. Examination of the patient while in hospital produced no evidence of disease apart from the condition of the leg.

The X-ray appearance was as shown in Figs. 318, 319. On a tentative diagnosis of myeloma, it was decided to perform a subperiosteal excision and await the pathological report before proceeding to more drastic measures.

Subsequent to the operation the films were shown to Dr. Gordon Thomson, Radiologist to Hounslow Hospital, who was absent from the department during the month of September. He has supplied the following description of the radiological appearance of the leg :—

“ There is a tumour which appears as a single lesion in the lower end of the shaft of the tibia. It extends along the medullary canal and has not invaded the epiphysis. The cortex is broken through, reduced in width, and not bulging. In the lateral view, the process of cortical destruction appears to be taking place from outside, rather than from within, the bone. There is some periosteal new bone formation in the tibia, and evidence of periosteal new bone on the outer side of the fibula.

"The X-ray appearance suggests a malignant tumour invading and destroying the bone. There are indications that the tumour may have started on the surface of the bone."

OPERATION (Sept. 17).—Assisted by Mr. Grant Batchelor, I performed a subperiosteal excision of about four inches of the lower end of the tibia. The skin and subcutaneous tissues were normal; the tumour was entirely subperiosteal, and the periosteum was intact but thickened. The bone



FIG. 318.—Antero-posterior view.



FIG. 319.—Lateral view.

was deficient posteriorly over an area of about two inches of the middle part; after removal the lower end broke away. The tumour was soft, greyish in colour, and moderately vascular; it extended downwards almost to the lower epiphysial line, and upwards along the medullary canal. There was considerable sclerosis of the shaft of the bone at the upper limit of the growth, where the medullary canal was almost obliterated.

After the receipt of the pathological report the patient was carefully re-examined for evidence of new growth elsewhere in the body, the skull and

jaws were examined radiologically, and the Wassermann reaction was done. The results of all these investigations were negative.

Convalescence was uneventful. The wound healed by first intention, and the patient left hospital at the end of three weeks with the leg in a plaster-of-Paris splint. Radiograms taken on Nov. 3 show active subperiosteal regeneration of bone. It is proposed to give deep X-ray exposures to the leg with a view to inhibiting local recurrence.

PATHOLOGICAL EXAMINATION.

(L. M. HAWKSLEY)

MACROSCOPIC APPEARANCE OF THE SPECIMEN.—A 4-in. segment of long bone, without periosteum. The upper extremity, which has been chiselled to a blunt point, is intact for $1\frac{1}{4}$ in. but shows no medullary cavity and is of densely hard consistence. A thin shell of compact bone forms half of the circumference of the remaining portion, the other half being largely eroded, with sharply-pointed, undetached fragments of bone extending downwards for 1 to $1\frac{1}{4}$ in., but deficient in the lowest part.

The cancellous portion of all but the extreme upper part of the bone is entirely replaced by slightly lobulated, finely papillary masses of greyish-white, soft, friable tissue, in which on section are seen numerous minute cysts, some containing clear, colourless fluid, others blood. An attempt made to bisect the specimen longitudinally had to be abandoned, owing to the readiness with which the central soft tissue became detached from the bone.



FIG. 320.—Showing general appearance of the epithelial tissue in a fibrous-tissue stroma containing giant cells. ($\times 55$.)

MICROSCOPICAL EXAMINATION.—In all the sections prepared from portions of the bone with the tumour tissue on its inner aspect, and also from the soft central growth alone, the histological appearances are similar (Figs. 320–322). The outstanding feature is the presence of an infiltrating epithelial



FIG. 321.—Showing multinucleated giant cells in the stroma and cysts in the epithelial network. ($\times 55$.)

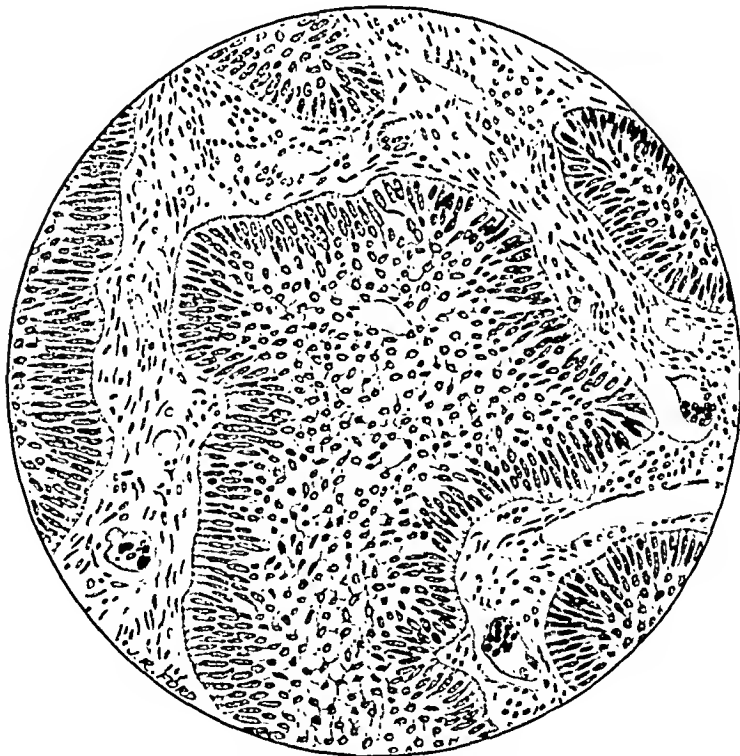


FIG. 322.—Detailed structure of the epithelial tumour, showing stellate cells (representing enamel pulp) with outer layer of columnar cells (representing ameloblasts); two small cystic spaces are seen in the epithelial network. The fibrous-tissue stroma contains multinucleated giant cells, suggesting the epulis type of adamantinoma. ($\times 200$.)

PRIMARY ADAMANTINOMA OF THE TIBIA 419

neoplasm in a fibrous-tissue stroma containing multinucleated giant cells. The appearance of the epithelial tissue at first sight recalls that of rodent ulcer, the growth being formed of masses of small cells arranged in sheets with irregularly projecting processes, and also in narrower strands, with an outer palisade layer of columnar cells; small foci with cystic change simulate gland lumina. The free border of the lobules of new growth is characterized by papillary formation of tissue of the same type.

Epithelial Elements.—

1. *Central cells.*—The centrally-placed cells consist of a network of small spindle-shaped and polyhedral cells with relatively large nuclei and radiating, delicate cytoplasmic processes, which anastomose with those of adjacent cells, the points of communication frequently being marked by a slight thickening. An occasional small whorl of the stellate cells, having a tendency to a more cuboidal form, is also seen. No 'epithelial pearls' or prickle-cells are evident in any of the sections. Spaces of varying sizes, ranging from narrow intercellular clefts to well-formed cysts, are present here and there in the epithelial network; some are devoid of contents, others contain coagulum, while in others there are strands of fibrin with enmeshed blood-corpuscles.

2. *Outermost cells.*—Surrounding each aggregation of anastomosing cells is a single layer of closely-packed, tall columnar cells with terminal bars, which form a continuous, sharply-defined border in contact with the adjacent fibrous-tissue stroma, the less clearly defined ends being directed to the central mass of stellate cells, a layer of cells of intermediate type frequently intervening. The large oval nuclei lie at that part of the cell farthest from the connective tissue; the nucleoli are not conspicuous. An occasional cell undergoing mitotic division is seen.

Stroma.—The stroma consists in the main of fibroblasts, with numerous multinucleated giant cells, whose nuclei number from three or four to thirty or more; in small areas there is more mature fibrous tissue, which has undergone extensive hyaline change. Interstitial hæmorrhage is a prominent feature in some of the sections, and in most of them macrophages containing granules of hæmosiderin are to be found. Isolated spicules of bone are scattered through both the central and the peripheral parts of the masses of tumour tissue. The stroma closely resembles myeloid epulis and benign giant-cell tumour, but there is evidence in many parts that the giant cells function as osteoclasts. Blood-vessels are few, but well-formed. No cysts are found in the stroma, and there is nowhere any indication of sarcomatous change.

Bone.—Sections of decalcified bone from the middle portion of the specimen show only a thin layer of compact substance, its inner aspect encroached upon by the tumour tissue.

DIAGNOSIS.—The epithelial new growth in this specimen has the histological characters of adamantinoma, the stellate cells representing the enamel pulp, and the columnar cells the ameloblasts, of the embryonic enamel organ. In none of the sections is there evidence of enamel formation.

In view of the non-metastasizing properties of adamantinoma of the jaw, and the absence of any history or clinical findings of any other neoplasm in the patient from whom this specimen was removed, the case must be regarded as one of primary adamantinoma of the tibia.

COMMENT.

The tumour is apparently identical with that recognized in the jaw under the names of 'adamantinoma', 'adamantoblastoma', and 'epithelial odontome', the enamel-forming elements of the developing tooth of a four-months' fœtus being represented in their characteristic arrangement. The pseudo-gland formation in the tumour is also a feature of the histology of the developing tooth.

The tumour possesses the power of eroding hard bone, a property which it shares with infiltrating neoplasms, e.g., rodent ulcer, derived from the basal cells of the epithelium.

Although it is recognized that adamantinomata occasionally arise in the region of the sphenoid, i.e., in bone only indirectly connected with the jaw,

it is difficult to put forward any view as to the manner in which an epithelial neoplasm differentiated so definitely along the lines of the embryonic enamel organ originated deep to the periosteum of the lower end of the tibia.

The only case of primary adamantinoma of the tibia which we have been able to trace in the literature—and it presents a close parallel—is that recorded by Bernhard Fischer¹ in 1913:—

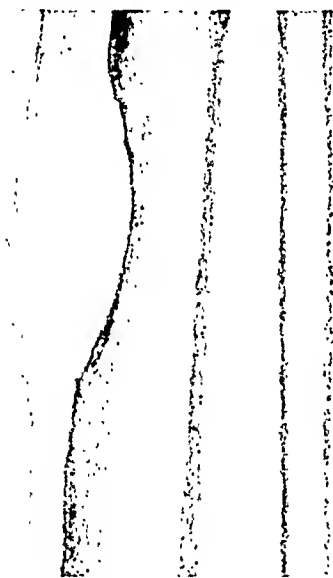


FIG. 323.—Radiogram of Fischer's case of primary adamantinoma of the tibia, lateral view. Compare excavated appearance of the eroded bone with that shown in Fig. 319. (By courtesy of the 'Frankf. Zeits. f. Pathol.')

A healthy man, age 37, slipped on an iron staircase and struck his left leg against one of the steps. Severe pain and bruising resulted, but passed off in a few days. Five months afterwards at the site of the injury he noticed a swelling, which, treated by local applications, increased in size and became very painful. On examination four months later there was found between the middle and lower thirds of the tibia a smooth swelling, nearly the size of a hen's egg, and tender on palpation. Radiological examination (Fig. 323) showed the presence of a tumour at this site. Resection of the involved portion of bone was performed and an osteo-periosteal graft from the sound leg inserted. Eight

months after the operation the patient was in good health and free from evidence of recurrence. Microscopically the tumour presented the characteristic picture of adamantinoma, with a spindle-celled stroma which in parts had undergone myxomatous degeneration.

Fischer, in discussing the mode of origin of the growth, discounts the possibility of a cell-rest composed of true enamel organ germ; and also of the transference of developing enamel organ from the oral cavity of the embryo to its tibia ("Man müsste denn annehmen, dass der Embryo zu der Zeit, als die Schmelzkeimanlagen entstanden, intrauterin in sein linkes Bein gebissen hat und dadurch einen Schmelzepithelkeim in die Tibia hinein befördert

hat. Aber diese Annahme dürfte zu absurd sein, um näher diskutiert zu werden.") In his opinion the condition could be accounted for by an excessive development of the multipotent embryonic ectoderm of the leg, with, in early embryonic life, its differentiation in the direction of enamel formation.

In the present case the epithelium must almost certainly have been subperiosteal from embryonic life: it would be difficult to account for its deposition subsequently by means other than a cutaneous wound, with implantation of a fragment of skin, or metastasis from a primary tumour elsewhere; of these there is no evidence. The possibility that the tumour might be teratomatous in nature would seem to be excluded by the entire absence of tissues other than those described above. The epithelium presumably remained of primitive basal-celled type, stimulated into tumour growth by the first injury, the second injury merely drawing attention to the presence of the condition by the onset of pain. For an explanation of such tissue dystopia we can only look to the embryologist.

REFERENCE.

- ¹ FISCHER, B., "Ueber ein primäres Adamantinom der Tibia", *Frankf. Zeits. f. Pathol.*, 1913, xii, 422.

**THE COLLOID ADENOCARCINOMATA OF THE
BLADDER VAULT ARISING
FROM THE EPITHELIUM OF THE URACHAL CANAL:
WITH A CRITICAL SURVEY OF THE TUMOURS OF THE URACHUS.**

By R. CAMPBELL BEGG,
GENITO-URINARY SURGEON AT WELLINGTON HOSPITAL, N.Z.

INTRODUCTION.

It is little recognized by surgeons, pathologists, or even urologists that tumours may occur at the bladder apex which, while presenting themselves clinically and cystoscopically as simple epitheliomata or papillomata, display when examined microscopically many of the characteristics of adenocarcinomata of the rectum. These neoplasms have as a rule undergone colloid—or, better, mucoid—degeneration, but still show clearly ducts and acini containing mucin and lined by columnar epithelium interspersed with so-called goblet cells. This phenomenon has often puzzled both the pathologist and surgeon concerned. The condition is usually explained by a vague reference to embryonic rests of the 'allantois' or urachus, after the preliminary hypothesis of metastasis from the bowel has been found untenable. Sufficient material is now available on the clinical side to show that these tumours as described form a very definite entity, being all similar in their development and histological characteristics. The question of etiology may be largely solved by a more exact study of the anatomy and pathology of the urachus. This enables us to follow the origin and progress of the neoplasms in a regular and progressive way seldom attained in such a study.

The subject will well repay consideration. It gives a remarkable insight into the potency residing in primitive cells, and the conditions under which they take on malignant characteristics. It is, therefore, not without value in the study of cancer in general. On the other hand, it is by no means devoid of practical importance. Adenoma or adenocarcinoma is probably the commonest tumour of the bladder apex. In addition to the personal one to be described, seventeen definite cases have been collected from the literature, many of them with such good and full descriptions and illustrations that the structure, clinical march, diagnosis, and treatment of the neoplasms can be laid down with exactitude. These cases, scanty in number, represent in no way the frequency of the disease. Several personal communications of unpublished cases have been received from medical confrères. It is remarkable how the publication of a hitherto unexplained phenomenon will bring an increasing number of reported cases in its train. The importance lies in the consideration that what presents itself under the guise of a bladder tumour is in reality not a bladder tumour at all, and any attempt to treat

it as such by the usual methods—fulguration, radium, etc.—is predestined to failure. The early recognition of the true condition offers the patient at least a hope of effective cure by surgical means.

The plan to be followed in this paper will be in *Part I* to fix the reader's attention by the report of a typical case at a fairly advanced stage, to consider briefly the anatomy of the tract involved, and to follow this by a study of the neoplasms as a group; and in *Part II* to give the results of some studies of the pathology of the urachus, in order to explain the etiology of the growths and to classify them.

It should be remembered that no part of the bladder has an absolute monopoly of any particular pathological condition. The twofold origin of the viscus and its epithelium from mesodermal (Wolffian) and entodermal elements is only faintly reflected in its pathology. Papillomata of the ordinary type may appear, though rarely, at the apex; and adenomata or adenocarcinomata with or without mucin formation or degeneration may occur in any part of the organ without relation to the urachus. A true tumour of the urachus does not necessarily appear at the site of this structure's insertion into the bladder. The growth may have tracked along outside the mucosa and appear on the posterior wall and eccentrically. This is rare, and such a case cannot be accepted as a urachal tumour unless its continuity with and origin from the urachus can be demonstrated. On the other hand, the studies of Störek and Zuckerkandl,^{115, 116} Albarran,³ and others have clearly shown the metaplastic potentialities of vesical epithelium, which can pass through all the stages of cystitis cystica to that of fully formed colloid adenocarcinoma (*see Fig. 339*). Indeed, in many cases the theory of urachal or allantoic rests has been unnecessarily dragged in to elucidate phenomena explicable along other lines. One case, not yet reported, of colloid cancer of the base of the bladder occurred in my practice. It was accompanied by so-called pseudomyxoma peritonei. The latter feature was no doubt unique, but the primary entity can easily be paralleled in the literature.

So definite, however, are the characteristics of apical vesical tumours of urachal origin, that there is no difficulty in differentiating them from the colloid cancers of the bladder wall itself, even when the latter occur towards the apex. The case of Kaltenbach⁶³ illustrates this point. The pedicle of a huge mass of tumour, resembling in microscopical structure a colloid cancer of the rectum, arose from near the apex. The structure of this growth was typically that of a bladder adenocarcinoma, arising from the mucosa and definitely pedunculated—a characteristic foreign to the urachal variety, which commences in the muscular wall and only subsequently penetrates the vesical cavity.

PART I.

CASE REPORT.

Mrs. B., female, age 54. Married, three children. Examined on Feb. 13, 1924. History.—For ten years has had intermittent attacks of scalding and pain on micturition, with frequency. These symptoms have been more continuous for the last six months. Recently two attacks of hæmaturia without clots. Feeling of pain and 'bearing down' in the region above the symphysis pubis. General condition good. Has not lost weight or appetite.

ON EXAMINATION.—

General.—Well-nourished, healthy-looking woman. General examination elicited nothing of note.

Local.—Slight dullness above the symphysis pubis for one inch, in and close to the middle line. Tenderness and a sense of resistance on deep pressure downwards and backwards towards the bladder. No tumour palpable. No cord palpable between the navel and bladder.

Vaginal Examination.—The bladder was tender. Nothing definite to be felt.

Urine.—Hazy, stale, and offensive, of the type rarely found except in cases of diverticulum or tumour of the bladder. Red blood-cells, pus cells, and staphylococci with *B. coli*.

CYSTOSCOPIC EXAMINATION.—Mucosa generally inflamed, but not so as to obscure the vessels. At the apex and slightly excentric towards the left side was a shallow ulcer, circular, and $1\frac{1}{2}$ cm. in diameter (Fig. 324). The surface was irregular, reddish

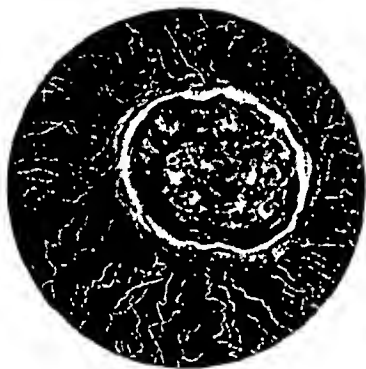


FIG. 324.—A cystoscopic view of the tumour.

on the whole, but with some white necrotic material in the centre. The edges were slightly raised, but did not appear to be indurated. There was no underenting. The ulcer bled freely at the touch of a ureteral catheter and was painful. A piece was removed from the edge for pathological study.

Pathology of Tissue Removed.—

"Tissue is highly vascular. The stroma is diffusely infiltrated with indefinite acinar forms. The cells lining the acini are of the high cylindrical type with nuclei at the base. Many are of the goblet variety. The tissue resembles an adenocarcinoma of the rectum" (Dr. Lynch).

The usual methods were used to exclude the possibility of a primary growth elsewhere.

OPERATION.—Before handling the bladder the peritoneum was opened from the umbilicus downwards and the whole abdominal cavity and bowel searched by touch and vision. Nothing abnormal was found. The urachus was isolated and appeared to run into a small, hard nodular growth at the bladder apex. The peritoneum was tacked down to this growth behind and was whitish and fixed, but the tumour had not ruptured through it. The whole mass, including the urachus and all the tissues between the transversalis fascia and the peritoneum, was removed together with the upper half of the bladder.

AFTER-HISTORY.—Quick convalescence. All urinary symptoms except frequency due to the reduced size of the bladder disappeared.

Eighteen months after the operation the patient was examined. The bladder had reached normal capacity. The urine was amber-clear. There were no pathological elements. Faint scar detected with the cystoscope at the site of resection. Nothing to be felt in the suprapubic region or pelvis. No urinary disturbance or complaint of any kind.

Two and a half years after the operation the bladder was still normal. There was no urinary trouble. A mass could be felt in the pelvis by bimanual examination just inside the left vaginal fornix.

Four months later during my absence she consulted a surgeon who, unfamiliar with the previous history, opened the abdomen and found the whole space between the transversalis fascia and peritoneum filled with recurrent growth. The glands of the pelvis on the left side were also involved. He contented himself with removing a piece of the tumour for examination. The patient died a few months later, almost

three years after the operation, without having had any recurrence in the bladder itself.

SPECIMEN REMOVED (Fig. 325).—The tumour is cone-shaped, covered on one aspect with peritoneum, and attached to and perforating the bladder wall, forming there a circular ulcer between 1 and 2 cm. in diameter. A ring of the complete wall of the bladder, including mucosa, is attached to the specimen, giving a margin of several centimetres of healthy mucous membrane around the ulcer. On section there is seen a hard coagulated greyish jelly-like substance veined with pale red lines. Towards the lower part the appearance is more compact.

Microscopical Examination (Figs. 326, 327).—There is an indefinite muscufibrous condensation limiting the tumour above; below, the latter is in direct contact with the bladder muscle. There is a thin-walled stroma of vascular fibrous tissue throughout. Between the strands of this stroma are masses of irregular cell columns and acini. The latter are lined with a single layer of high cylindrical epithelium with basal nuclei. The acini are full of a homogeneous material that takes strongly a basic stain.

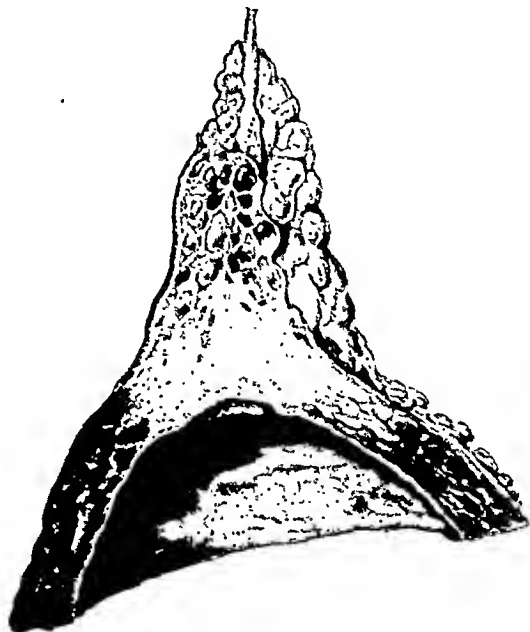


FIG. 325.—Semidiagrammatic view of the specimen removed, showing the ulceration of the mucosa of the bladder and the relation of the urachus to the tumour. The peritoneum and prevescical fat can be seen.

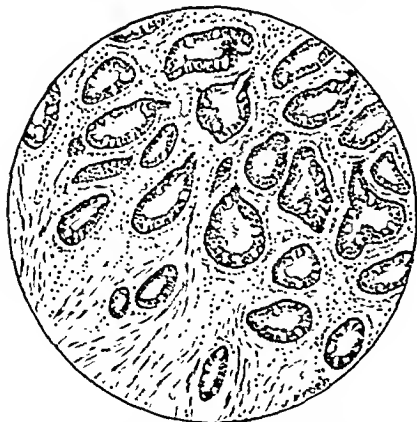


FIG. 326.—Low-power view of a section taken close to the bladder mucosa. The acini are in direct contact with the smooth muscle fibres.

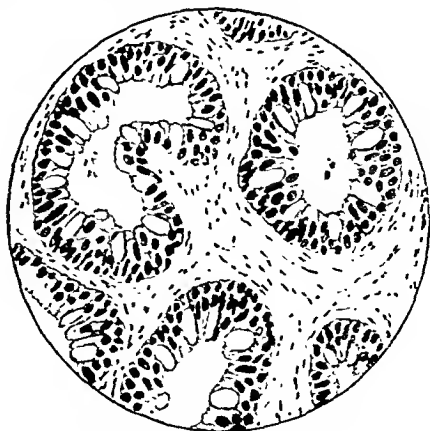


FIG. 327.—High-power view. Goblet cells are numerous, but the epithelium is not so regular as in rectal growths. Some of the columnar cells are based on a row of cubical ones.

There are many goblet cells and others with mucin granules. Towards the bladder mucosa the acini are perforating and invading the bladder musculature, and may be seen among the individual fibres. At the upper part of the tumour the acini formation is less distinct. Many of the cells have undergone complete degeneration, and lie in unlined spaces filled with mucin. This substance appears also in the meshes of the stroma itself. The tumour is a columnar-celled carcinoma resembling the type frequently seen in the rectum.

The piece of growth removed from the metastatic mass a few months before the patient's death showed a similar structure, but with less colloid degeneration.*

To avoid redundancy, a more detailed description of the microscopic structure is reserved until a general survey has been made of all the cases reported. Prior to that it is necessary to summarize briefly the anatomical features of the urachus. A complete study on this subject has already been published.¹²

THE ANATOMY OF THE URACHUS.

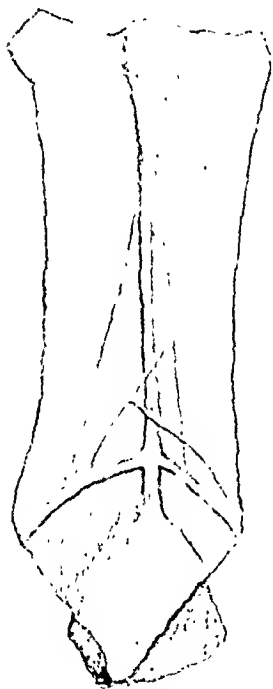


FIG. 328.—Drawing of the urachus and ligamentum commune of a child of three years as seen from the peritoneal aspect. The apex of the urachus is indicated by the point of junction of the umbilical arteries. (See also Figs. 349, 350.)

The tumours of the urachus at the bladder end, though frequently not recognized as urachal tumours at all, are by far the most important clinical group. To get a comprehensive view, however, it is necessary to consider the urachus and its adnexa as a whole, as faulty misconceptions on this subject have led many writers to draw unwarranted conclusions. The urachus lies in the space of Retzius. Considered in its widest sense, the latter embraces all that potential space bounded anteriorly from below upwards by the posterior and upper aspect of the symphysis pubis and the transversalis fascia; behind, by the bladder as far as its peritoneal reflexion, and above that by the peritoneum.

Ideally, though never actually, it forms an isosceles triangle, whose lateral boundaries are the obliterated umbilical arteries and whose base is formed by the puhoprostatic ligaments below. The apex of the triangle is the point where the two umbilical arteries and the urachus meet above. This point is, at birth, the umbilicus, but as the urachus descends immediately, it is much lower down in the adult, being situated as a rule at the junction of the lower with the middle third of the distance between the bladder apex and the umbilicus (Fig. 328, 329). As there is more or less union between the umbilical arteries and the two structures between which they lie—namely, the peritoneum

* The term 'colloid' is in such constant use that it is not possible to avoid it in connection with these growths. It must be remembered, however, that the material formed is, strictly speaking, mucin and not colloid.

behind, and the transversalis fascia in front—the space is closed laterally. Lower down it passes deeply over the sides of the bladder into the pelvis. With this portion, however, we are not concerned.

The urachus descends, and as it is attached on each side to the umbilical arteries, the latter are drawn together and form one continuous band passing from its apex to the umbilicus. The name of *ligamentum commune* has been suggested by Professor Peremeschko⁹³ for this structure (Figs. 328, 329, 331). It consists merely of the drawn-out adventitial muscular and possibly elastic tissue derived from the walls of the obliterated umbilical arteries. It may give rise to the mesoblastic tumours which when they occur in this region have been erroneously attributed to the urachus.

On the assumption that it normally extends as far as the navel, it has, indeed, been the fashion to attribute to the urachus almost any pathological condition occurring in the upper part of the space of Retzius. Tumours, however, may have their origin from any of the structures, fibrous, fatty, or vascular, above, or from the bladder itself below, and in both cases may involve eventually every part of the space, working down to and invading the bladder from above, or passing up from it towards the umbilicus. With many of these, the urachus has no connection.

Divergences from the Ideal Anatomical Relationships.—It must be taken into consideration that the actual anatomical conditions which prevail are not the ideal ones described above.

There is as a rule, it is true, the central *ligamentum commune*, but frequently the umbilical arteries are so teased out by the descending urachus that innumerable fibres of their adventitia are spread in a fan-shaped manner, sometimes over an area as wide as three inches (Figs. 328, 330). One or both of the umbilical arteries may be avulsed completely and lie free, being only connected to the umbilicus by fine strands of adventitial tissue. Thus, instead of one space, many potential ones exist, bounded by the strands of this so-called Luschka's plexus. Again, the urachus is frequently not central. It may have lost its attachment to one umbilical artery, and its upper end may be pulled laterally and at the same time in a downward direction by the other, so that it takes a course from the bladder apex upwards and outwards to the right or left (Fig. 330). *Urachal tumours are accordingly not always in the middle line.* Even where it is laterally displaced, however, the urachus is firmly attached by fibrous strands to the umbilicus so that it

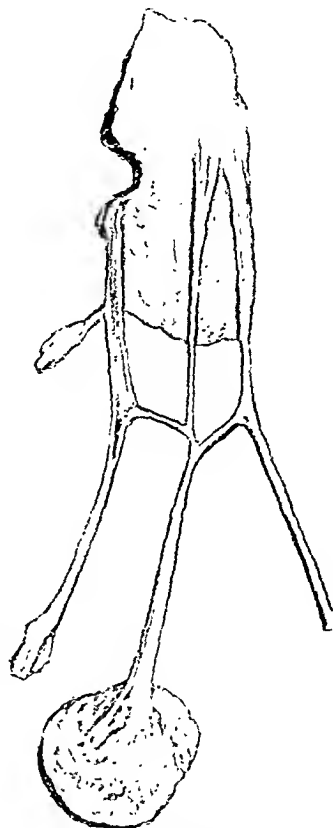


FIG. 329.—A dissected specimen of the urachus and attached ligaments from an adult. Note the splitting of the left umbilical artery.

can carry out its musculo-tendinous function of steadying the bladder during micturition.

Rests from Urachus left during its Descent.—In previous papers^{12, 13} I have suggested that the urachus during its descent may be pulled asunder or that a portion may be detached, thus leaving some of its cellular content behind, and that from these rests epithelial tumours may arise. These tumours may be found at the umbilicus itself, or between that structure and the apex of the urachus 10 or 11 cm. below. Certain adenomatous structures and neoplasms occurring in this region are referred to later (*see Figs. 349, 350*).

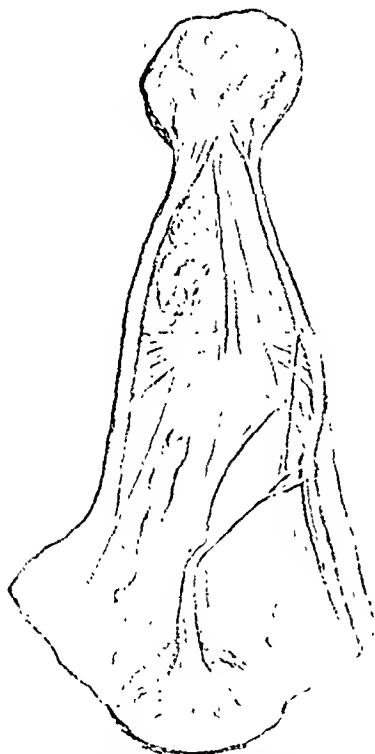


FIG. 330.—A drawing of an adult urachus to illustrate how the structure may be pulled over by losing its attachment to one of the umbilical arteries.

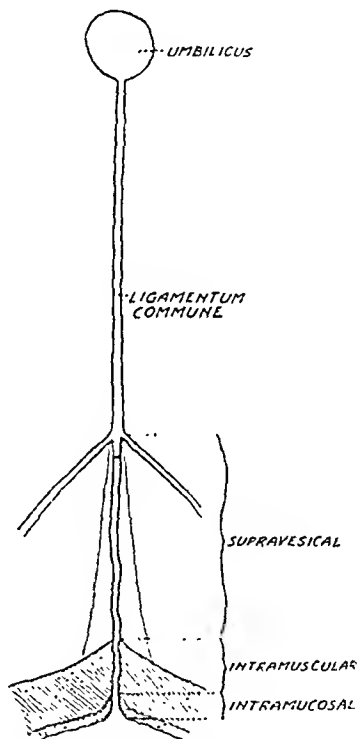


FIG. 331.—A diagram to show the subdivisions of the urachus.

Divisions of the Urachus.—The urachus is a structure about 5 to 6 cm. in length, and for our present purpose can be conveniently divided into three portions (*Fig. 331*): (1) Supravesical, where it is entirely above the bladder; (2) Intramural, where it lies in the muscular wall; (3) Intramucosal, in cases where its lumen is in direct communication with the bladder cavity. In the 66 per cent of cases where there is no communication, there are only two portions to be considered, namely, supravesical and intramural. In all three parts the canal is patent and lined by many layers of epithelial cells (*Figs. 332–335*).

FIG. 332.—Low-power view of a urachus. The specimen was taken from a male adult, 1.5 cm. above the bladder.

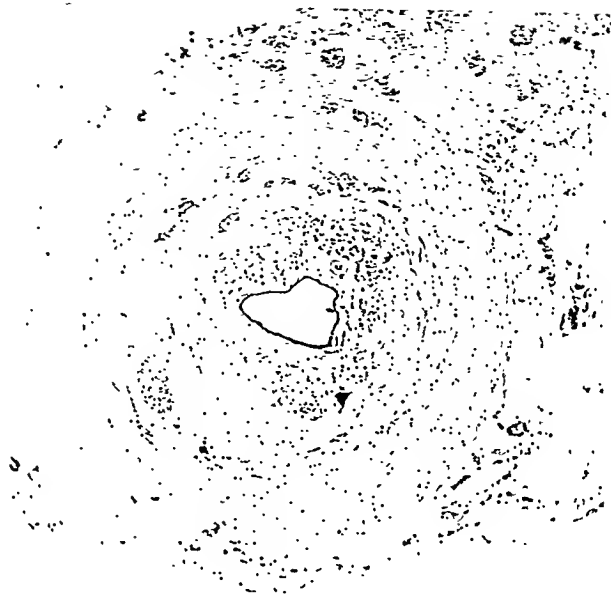


FIG. 333.—Section through the apex of the urachus, 5 cm. above the bladder. The epithelium forms a solid column.



FIG. 334.—Section of an adult urachus 2 cm. above the bladder. The irregular heaping up of the cells is well shown.

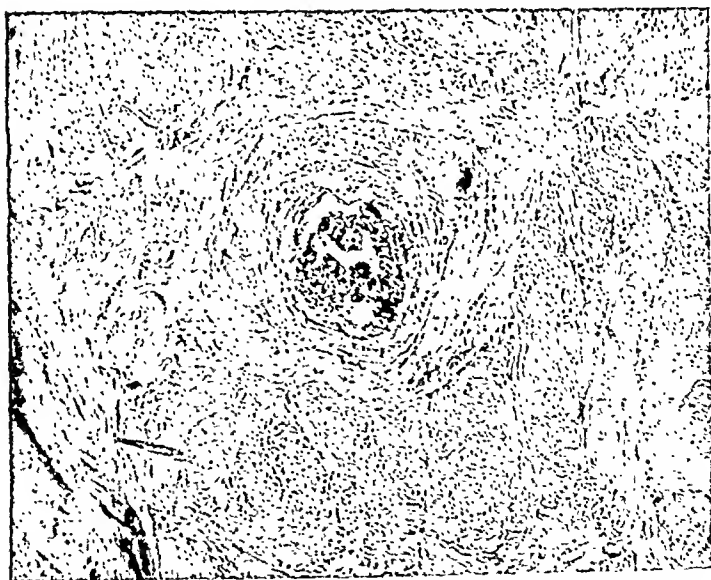


FIG. 335.—The same urachus as shown in *Fig. 334*, 4 cm. above the bladder.

That centimetre of the urachus which penetrates the muscular wall of the bladder is the most important from the pathological point of view, for it is usually at the upper part of this, or in the lower end of the extravescical section, that new growths originate (*see Figs. 337, 338, 340*).

In some cases where the canal communicates with the bladder there is a wide trumpet-shaped opening forming a diverticulum (*Fig. 336 A*). This probably existed in the cases of Michin,⁸³ Randall,⁹⁹ and Lavenant,⁷⁴ where the growths were seen to project from a funnel-shaped retraction of the bladder apex. In some instances the opening is at the top of a papilla (*Fig. 336 B*), and at others it is flush with the bladder mucosa. Neoplasms in the former would tend to project into the bladder, and in the latter appear as a flat ulcer. Both these types have been found.

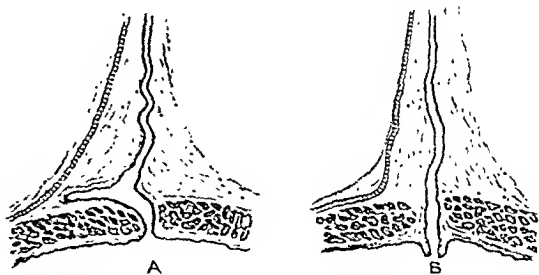


FIG. 336.—Two methods by which the urachus may connect with the bladder. In A there is a wide expansion; in B the canal opens on the summit of a papilla.

Unformed and Undescended Urachus.—In a previous paper on umbilical fistulae of urachal origin,¹³ it was shown: (1) That in some cases the urachus does not form at all—that is to say, the upper part of the bladder fails to narrow at the appropriate stage of foetal life, and as a result its apex remains at the umbilicus, no descent having taken place; (2) That in other cases the urachus is not properly differentiated from the bladder, the cavity of the latter being continued by a wide canal as far as the umbilicus. All intermediary stages of formation and descent may be encountered, the apex of the bladder reaching in some instances well above the symphysis. In these the urachus is usually normal, but placed at a higher level. This fact is of some importance, for a tumour which is really in the intramural part of the urachus may be palpated well above the symphysis, as in the cases of Michin⁸³ and Schwarz.¹¹⁰ Finally, in the extreme case of ectopia vesicæ, the urachus and the bladder remain open anteriorly and the apex of the combined organs is at the umbilicus.

CLASSIFICATION OF TUMOURS OF THE URACHUS.

Classification according to Position (*see Fig. 331*).—Tumours of the urachus may be divided into those:—

1. Of the intramucosal portion, where this exists (33 per cent of cases).
2. Of the intramural portion.
3. Of the supravescical portion, which extends upwards 5 or 6 cm. from the apex of the bladder.
4. Of abnormal urachal rests above the apex of the urachus and between this and the umbilicus.
5. Of an unformed or undescended urachus. In the former case the neoplasms, strictly speaking, belong to the upper part of the bladder.

6. Of the fibrous and muscular strands derived from the umbilical arteries above the true apex of the urachus. They are not tumours of the urachus at all, but it is convenient to specify them here, as many of the cases reported as urachal neoplasms belong to this group. Microscopically they contain no epithelial elements. They are fibromata, fibromyomata, or sarcomata.

7. Of the upper part of an ectopic bladder, which is in reality the anlage of the urachus.

Classification according to Type.—This classification is based on a study of all cases of neoplasm of the urachus found in the literature. A large amount of sifting of material has been necessary to exclude those reports where the evidence was against the urachal origin of the neoplasm described. On the other hand, many probable urachal tumours lie buried under other captions. The help of current indexes is no doubt inadequate to disinter some of them, so that it is improbable that the list given is complete; it provides, however, sufficient material for a satisfactory analysis. One variety of neoplasm calls for special mention. In the absence of undescended urachus, tumours derived from urachal rests may form at the umbilicus itself. Apart from this theoretical possibility, some of the actual growths in this region resemble so exactly proved urachal tumours that there is little doubt as to their true nature. The whole subject of adenomatous growths of the umbilicus is, however, so extensive that its inclusion would complicate unnecessarily the problem to be considered. Apart from casual mention and a limited reference to the literature in the bibliography, it will not be dealt with (*see* p. 461).

Rejection of certain Cases from the Category of Urachal Growths.—Six cases should, I think, be excluded from any future statistical study of the subject. As they are repeatedly used to illustrate urachal pathology, short notes and comments are given of each of them. The case of Pinheiro⁹⁶ said by Kiellenther to be one of carcinoma of the urachus was not available to me in the original, and is accordingly not included in this study. The other six are as follows:—

1. KLOPP'S case.⁶⁷

Klopp described in 1921 as sarcoma of the urachus a case (male, age 50) in which a small collection of mononuclear cells accompanied by a giant cell were found in connection with a canal lined by squamous epithelium discharging at the umbilicus. The evidence that this was a tumour of the urachus, or indeed a tumour, at all is unsatisfactory, though the fact that the sinus reached down as far as 12½ cm. from the umbilicus might relate it with the apex of the true urachus. Mononuclear and giant cells have been found in simple cases of umbilical fistula.

2. RANKIN and PARKER¹⁰⁰ (Case 7 in reference cited).—

This was apparently a mixed tumour of small size in a male, age 68. It was a squamous-celled carcinoma with a few irregular spaces lined by epithelial cells. It was said to be attached to the urachus, which was discharging at the umbilicus. It is, however, very easy to confuse the thick strand of the ligamentum commune with the true urachus. As is well known, most of those interesting cases with a pus discharge at the umbilicus do not concern the urachus at all, and merely track down for a few centimetres in the abdominal wall. In the same article the authors mention three other undoubted cases of urachal tumour.

3. GÖBEL'S case⁴³ (published in 1905).—

Male, age 40. A cancer, originating in a lesion caused by *Schistosoma hamatobium*, had perforated the anterior wall of the bladder and finally ruptured through

the abdominal wall at the umbilicus and at another place farther down. All tumours extending in the space of Retzius, however, tend first of all to rupture through the skin at the weak point represented by the umbilical scar. The apex of the urachus is normally 11 cm. below this, and there seems to be no reason to think that this structure was involved. The case was published as one of a very long series in which carcinoma had supervened on bilharziasis. In the main, the growth had the characteristics of a squamous carcinoma.

4. HOFFMANN's case⁵⁷ (published in 1870).—

Male, age 28. This was said to be a carcinoma in a patent urachus involving the bladder. A careful study of the case shows that the bladder had never left the umbilicus and the urachus had not formed. The narrowed apex of the bladder reached the umbilicus, and starting from this point the organ became involved in a squamous epithelioma. There is no evidence that this was primary in the bladder, and it may easily have originated from the deep surface of the umbilicus, from ectodermal elements included in the umbilical scar at the time of its formation, or from some other rest in the neighbourhood. Possibly it arose from the vesical mucosa, as squamous epitheliomata are not very uncommon in the bladder.

5. FISCHER's case³⁶ (published in 1893).—

Male, age 32. The patient is said to have had a patent urachus till the age of three, with urine discharging at the umbilicus. A carcinoma developed in the region of the navel and ruptured through the skin. The nature of the carcinoma is not stated. At the autopsy it was found that the growth had involved but not penetrated the wall of the bladder at the apex. The latter was in the normal position. The urachus had thus formed and descended, and the case was probably one of carcinoma arising in the neighbourhood of the umbilicus and involving the bladder by direct downward spread in the space between the peritoneum and the transversalis fascia.

6. LONG's case.⁷⁸

Long published a case in 1927 in which he found some hairs in a cyst at the umbilicus. He described it as a dermoid cyst of the urachus. The evidence produced in support of this diagnosis is not completely convincing.

Adenocarcinoma and Colloid Cancer Arising in the Upper Part of Ectopic Bladders.—As already pointed out, the upper part of the ectopic bladder is the anlage of the urachus, and reference to adenomatous structures in that region is of passing interest.

Six cases at least have been reported. In those of Zuckerkandl,¹³⁴ Ehrlich,³² and Hager,⁴⁸ the colloidal element predominated, while those of Scholl¹⁰⁸ (Case 5 in reference cited), Gerahty,⁴⁰ and Störck^{115, 116, 34} were simple adenocarcinomata of the intestinal type.

ANALYSIS OF CASES OF URACHAL TUMOURS IN REGARD TO TYPE AND POSITION.

Including the author's, 44 cases are given in the table on p. 434. Ten of these are mesoblastic tumours which, though not considered to be urachal neoplasms, have been published as such. The 44 are divided as follows:—

Fibro-adenoma	3
Adenocarcinoma (not colloid)	4
Colloid adenocarcinoma	19
'Mixed tumours' (including those of unusual pathology)	8
Fibroma and fibromyoma	4
Sarcoma	6
Total	44

The determination of the nature of the cases does not rest entirely on the individual author's opinion, but on a very careful study of the individual descriptions and a comparison of these with each other, having in mind the true anatomy, morbid and systematic, of the urachus. The reports cover a fair period of time, and vary considerably in completeness. It appears probable that fibro-adenoma, adenocarcinoma, and colloid cancer result progressively from certain changes in urachal cells which can be studied in a large series of specimens taken from the post-mortem and dissecting-room, and they are accordingly grouped as one series.

The term 'mixed tumours' denotes a group presenting unusual features. They contained in some instances both sarcomatous and carcinomatous elements, and had a tendency to cystic or acinar formation. They were all connected with the apex of the bladder, and were in some instances called teratomata and attributed by the authors reporting them to 'allantoic' rests.

The ten cases of fibroma, fibromyoma, and sarcoma arose from non-epithelial structures, and when the position of the tumours is analysed it will be seen how strikingly this class is confined to the region of fibrous bands and teased-out arterial adventitia above the level of the urachus. There is of course no reason why such tumours should not arise from the same elements in the urachus itself, but as a matter of experience urachal tumours seem invariably to contain epithelial elements.

The table annexed gives an excellent impression of the situation of the different examples.

CASE INCIDENCE OF DIFFERENT VARIETIES OF URACHAL TUMOURS.

CLASS OF TUMOUR*	COLLOID CANCER	ADENOCARCINOMA	* MIXED TUMOURS*	FIBRO-ADENOMA	SARCOMA	FIBROMA AND FIBROMYOMA	TOTAL
I, II	5 (a)	2 (d)	1 (g)	1 (k)	—	—	9
III	13 (b)	1 (c)	6 (h)	2 (l)	—	—	22
IV	1 (e)	1 (f)	1 (i)	—	—	—	3
V	—	—	—	—	—	—	0
VI	—	—	—	—	6 (m)	4 (n)	10
Total	19	4	8	3	6	4	44

*I = Intramucosal. II = Intramural. III = Supravescical. IV = Tumours derived from urachal rests. V = Tumours arising in an unformed or undescended urachus. VI = Tumours not involving the bladder and arising from adventitial and muscular structures in the space of Retzius.

Key to reporters of cases indicated in table:—

a. Author's case, Kiehlentner,⁶⁶ Zuekerkandl,¹²⁴ Scholl¹⁰⁸ (Case 4), de Korte.²⁹ b. Chute and Crosbie,²⁶ Störck and Zuekerkandl¹¹⁶ (two cases), Lavenant,⁷⁴ Barringer,¹⁰ Schwarz,¹¹⁰ Randall,²⁹ Green,⁴⁵ Scholl¹⁰⁸ (Case 3), Rankin and Parker¹⁰⁰ (Case 5), Khaun,⁶³ Michin,⁶³ Pendl.³³ c. Hue and Jacquin.³⁹ d. Hartmann,⁵⁰ Scholl¹⁰⁸ (Case 1). e. Scholl¹⁰⁸ (Case 2). f. Koslowski.⁶⁹ g. Blavet di Briga.¹⁵ h. Rotter,¹⁰³ Rankin and Parker¹⁰⁰ (Case 6), Lenormant,⁷⁷ Nuboer,¹⁹ Cullen.²³ Doiran.³⁰ i. Adam.¹ k. Hagner.⁴⁹ l. Nicholson,⁸⁷ Hector.⁵² m. Greig.⁴⁶ Frank.³⁸ Villa.¹²² Greig Smith (two cases),¹¹³ Vönelken and Cambresier¹²⁵ (Case 2). n. Brady,¹⁸ Rankin and Parker¹⁰⁰ (Case 4), Aveling,⁹ Vönelken and Cambresier¹²⁵ (Case 1).

Remarks on the above Table.—*Class VII*, relating to growths in cases of ectopia vesicæ, is not included. Under *Class V*—tumours in an unformed urachus—not a single example occurs. Hoffmann⁵⁷ and Fischer³⁶ were inclined to place their cases under this heading. They have been referred to above and rejected from the category of urachal neoplasms.

No distinction is drawn between intramucosal and intramural urachal growths. The former would be expected to be rare, as from the anatomy of the urachus they could only occur in 33 per cent of the total—the percentage of cases in which the urachus communicates directly with the bladder cavity. Such growths would appear as bladder tumours and be mistaken for them, just as a growth projecting into the duodenum from the ampulla of Vater might commence so near to the mucosal junction as to be indistinguishable on the basis of situation alone from a tumour of the duodenal mucosa itself. Kielleuthner⁶⁶ thought that his case came under this category, but a study of his description and illustrations shows the origin of the tumour to have been intramural.

The distinction between the intramural and supravescical tumours is a real one, the pathology and symptomatology being different to some extent. The line is, however, hard to draw between borderline cases. The early malignancy and rapid onset of severe bladder symptoms characterize the intramural variety. The determination was made by considering the march of the symptoms and the nature of the growth as found at operation or autopsy; also the evidence of the age of different sections estimated from the advance of degeneration. The name of the author is given as sufficient indication to enable the reader to locate in the bibliography the report where the full description of the case will be found. These cases vary from the least advanced one of Kielleuthner,⁶⁶ in which the tumour was confined to the bladder wall, to that of Pendl,⁹³ in which a huge growth occupied a large part of the space of Retzius and reached almost to the umbilicus.

The preponderance of the colloid type over all other varieties, when one considers merely those tumours which arose from the part of the urachus within the bladder wall or immediately above, will appear from the following figures of recorded cases :—

Colloid adenocarcinoma	18
'Mixed tumours' of unusual pathology	5
Adenocarcinomata without colloid degeneration	3
Fibro-adenomata (2 discovered incidentally on post-mortem examination)	3
				<hr/> 29

Not a single one of the cases penetrating the bladder mucosa was of the mesodermal type.

Pathological Study of Colloid Carcinoma of the Urachus involving the Bladder Apex.—With the exception of those of Störek and Zuekerkandl, all the eighteen cases referred to under (a) and (b) in the table on p. 434 were reported between the years 1911 and 1930. The tumours are all essentially similar in microscopical appearance, and doubtless arose from the tiny adenomatous structures which are such a striking feature of the lower end of

the urachus as studied in routine post-mortem specimens (*Figs. 337, 338*). Many of the fine details described will become significant when compared with the latter. The growths found clinically represent the expected end-results of processes observable in the apparently normal urachus.

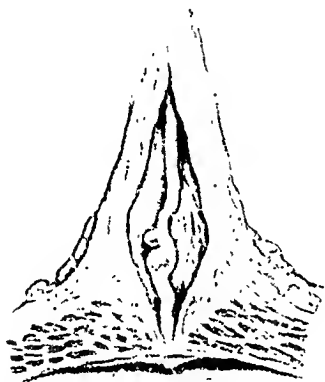


FIG. 337.—The urachal canal dissected from its sheath, showing two small cysts at its lower end.

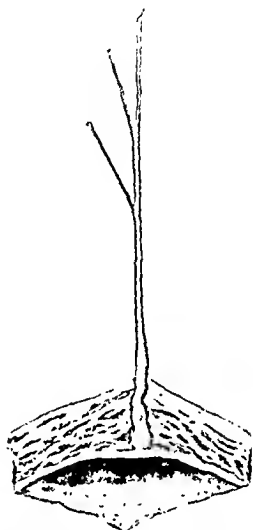


FIG. 338.—Urachus ending blindly at the mucosa, and dilated into a small cyst at this point. From a female 56 years of age.

Distinction between the Urachal Tumours and other Adenomatous Growths in the Bladder.—Lendorf⁷⁶ demonstrated that in the embryo there were no downgrowths of the mucous membrane to form glandular structures,

while in children these formations appeared as solid buds, and later in life as definite simple glands. In adults the latter may take on a more complex structure, and in cases of inflammation definitely secrete mucin, as exemplified in cystitis cystica. (*Fig. 339*.)

(Compare also the work on this subject of von Brunn¹²⁴ and Albarran.³)

The small multiple tumours present in cases of cystitis cystica become important pathological structures, and occur either singly or as multiple neoplasms arising from the bladder mucosa. These may take the form of simple adenomata, adenocarcinomata, or colloid growths. The structure of the latter may be similar to that of

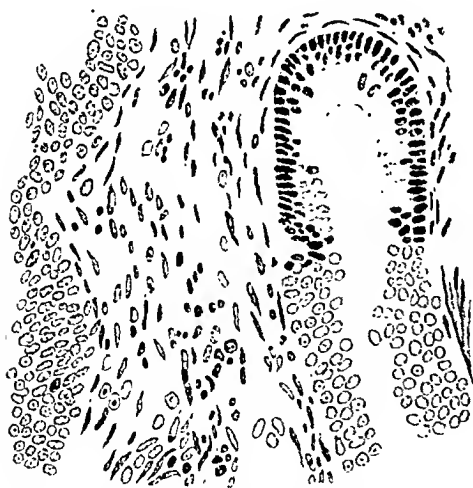


FIG. 339.—An illustration from Bayer's article to show the change of transitional epithelium into columnar. The section is taken from the bladder. It will be noted that the transitional epithelium has reverted to a primitive type (see p. 460).

colloid cancer of the rectum or to that of the urachal tumours which resemble it.

These pure bladder neoplasms may be differentiated from urachal ones in that they arise from the mucosa, tend to be pedunculated, and only penetrate the muscular wall in the advanced stage. The urachal tumours, on the contrary, originate at the apex of the bladder either in the muscular wall or completely outside it, and penetrate the mucosa later, or in a few cases merely bulge it.

Pure vesical adenomata of the simple type are exemplified in the cases of Albarran,³ Astraldi,⁷ Bridoux,¹⁹ Cahen,²³ Carraro,²⁴ Cassanello,²⁵ Egger,³¹ Herbst and Thompson,⁵⁴ Judd,⁶² Lavaux,⁷³ Motz,⁸⁶ Notthafft,⁸⁸ Pavone,⁹² Sacchi,¹⁰⁵ Watson,¹²⁹ and Uteau.¹²⁰ Instances of adenocarcinoma have been reported by Alexander,⁴ Audry,⁸ Bristow,²¹ and Kanamori⁵⁴; colloid cancers by Bayer,¹¹ Haake,⁴⁷ Kaltenbach,⁶³ Mandelbaum,⁸¹ Pinesohn,⁹⁵ Posner,⁹⁷ Rauenbusch,¹⁰¹ Rochet and Martel,¹⁰² Sauter,¹⁰⁶ Schmidtman,¹⁰⁷ Sharp,¹¹¹ Sperling,¹¹⁴ Störck,¹¹⁵ Zausch¹³³ (two cases), Zuckerkandl,¹³⁴ and Fontana.³⁷ In addition to the reports of the above authors the subject is discussed in the writings of Aschoff,⁵ Blum,^{16, 17} Frisch and Zuckerkandl,³⁹ Gerahty,⁴⁰ Paschkis,⁹⁰ Verhoogen,¹²¹ von Limbeck,¹²⁶ von Brunn,¹²⁴ and Wägeler.¹²⁷

'Mixed Tumours' of the Bladder Proper.—The type of tumour we have called 'mixed' in the study of urachal cases has its counterpart also in neoplasms arising from the bladder epithelium. These growths have, as a rule, been described as sarcomatous, myosarcomatous, etc. They are characterized by the presence of spaces or acini lined by secreting columnar or cylindrical epithelium, and have frequently a cystic nature when considered macroscopically. Lenormant⁷⁷ in his article discusses them briefly. Other examples that may be referred to are described by Albarran,³ Clado,²⁷ Heinke,⁵³ Herezel,⁵⁵ and Krompecher.⁷⁰

Adenomata Occurring in the Trigone and its Neighbourhood.—The discussion of this type of tumour opens up the question of aberrant prostatic glands. Certainly the pathology is different from that of adenomatous growths in the fundus, though the two varieties probably overlap. Case reports and discussions on this subject will be found in the articles of Goodall,⁴⁴ Hermann,⁵⁶ Horn and Orator,⁵³ Lubarsch,⁷³ Paschkis,⁹⁰ Thomas,¹¹⁹ and Wittsach.¹³¹

SYSTEMATIC DESCRIPTION OF THE COLLOID ADENOCARCINOMA OF THE LOWER END OF THE URACHUS.

While all the eighteen cases cited are essentially similar in their microscopical structure, they may roughly be divided into two classes having regard to their clinical course, macroscopical appearance, and the exact method by which they originate.

Class 1.—This comprises the great majority, in which the upper part of the growth is encapsulated and extends well up into the space of Retzius, whereas the lower end which is in the bladder wall has no definite capsule, the acini lying in direct contact with the muscular tissue. Most of them have ultimately penetrated the mucosa, but the length of symptomless history as regards the bladder, combined with their large size and the presence of

calcification in the capsule and stroma, leads to the conclusion that they existed for a considerable time as simple tumours of the type described in the cases of Nicholson,⁸⁷ Hector,⁵² and Hagner,⁴⁰ and took on frankly malignant characteristics late in their course.

Class 2.—This group is represented in the cases of de Korte, Albarran, Kiellenthner, that of the author, and possibly Scholl's *Case 4*. The neoplasms in this category apparently arise from microscopic adenomatous structures of the urachus, and become malignant at a very early stage. They are almost entirely within the wall of the bladder, and form cone-shaped tumours (*see Fig. 325*) with the apex above. There is no true capsule—at most a condensation of the tissues around the periphery. In the cases mentioned the growths were first discovered by cystoscopic or post-mortem examination, and no tumour was palpable. As in Class 1, however, the most degenerated areas were always those most remote from the bladder mucosa, suggesting that the point of origin was the upper part of the intramural portion of the urachus. Their pathological appearance is similar to that of the lowest portions of the first group.

In a few instances of the cases described under Class 1, the mucosa had not been penetrated at the time of examination. These no doubt all occurred in subjects in whom the lumen of the urachus reached only as far as the external surface of the bladder mucosa (*see Figs. 337, 338*). Such are the cases of Barringer and Pendl, and Störek and Zuckerkandl's *Case 1*.

As found at operation and subsequent examination or autopsy, the growths ranged from the size of a hazel-nut in Kiellenthner's case to that of an adult head in Pendl's. Two portions could be distinguished, an upper involving the whole or part of the supravescical urachus, and a lower incorporated in the bladder wall at the apex.

The capsule was in direct continuity with the muscular and adventitial wall of the bladder below, and ended there. Within the musculature of the bladder wall the cells invaded directly the surrounding tissues, lying in apposition with the muscular fibres, in some cases penetrating their sheaths.

The general consistency of the tumours was cystic, and they were composed in every instance of cavities lying in a network of stroma. These cavities varied in size from large ones with several ounces of content to tiny spaces only distinguishable under the microscope.

The upper part of the tumours lay between the transversalis fascia and the peritoneum in the anterior abdominal wall, near to the middle line. They were all adherent to the peritoneum towards the bladder end, and could not be removed without opening the peritoneal cavity. As a rule they were easily shelled out after severing the ligamentum commune, which attached them to the umbilicus, down to the point where they were adherent to the peritoneum. In more than one instance the growth had broken through the peritoneum, and in one had formed adhesions with the abdominal contents. In Pendl's case the tumour was adherent also to the transversalis fascia and the recti muscles.

From the upper part of the growth a firm cord-like structure may pass up towards the umbilicus (*see Fig. 325*). It may on section show the epithelially-lined canal of the urachus which has been pushed up by the development

of the growth in a superior direction. If the canal is completely involved in the tumour, the only connection with the umbilicus may be by means of the ligamentum commune or the strands of Luschka's plexus (*see Figs. 329, 330*). In cases where the urachus has not descended, the growth may reach as far as the navel, but as a rule falls short of it even then.

The Capsule.—The capsule is always fairly definite, but may be markedly irregular, corresponding to the lobulated form of the tumour itself. It may be so thin that the gelatinous content shines through. It may be as much as 3 cm. in thickness, and consist of a layer of fibrous tissue covering one rich in smooth muscle fibres. In advanced cases the fibrous bundles are old, sclerosed, poorly supplied with blood, and may have undergone hyaline or calcareous degeneration. In some instances they harbour foci of inflammatory round cells.

The apparent capsule is not true capsule in its whole thickness, the presence of alveoli in the inner layer demonstrating that this portion is merely a compressed part of the tumour proper.

As mentioned already, the outer sheath of fibrous and muscular tissue becomes continuous with the adventitial and muscle layers of the bladder, so that the capsule is always incomplete below.

The Stroma.—The stroma consists of bands of varying thickness, and may be concentrated into a fibrous mass towards the centre of the tumour, as in the case of Barringer. It may show all the changes of hyalinization and calcareous degeneration, and in parts may actually be infiltrated by mucin derived from acinous groups at one time existing in the meshes. It encloses spaces of varying magnitude from small microscopical ones to huge cysts. In many cases the mucin in these spaces is in direct contact with the fibrous tissue, though there may be traces of the original cell lining. At the bladder end the stroma takes the form of young or medium fibrous-tissue bundles lying irregularly among the acini of the tumour and the muscle fibres of the bladder. The latter tend to be replaced by the fibrous tissue (*see Fig. 326*).

The Epithelial Element.—So great is the output of mucin that rapid degeneration both of the cellular elements and the stroma takes place, and the undegenerated portion or growing edge is small. In all the cases reported, however, that section of the tumour immediately deep to the bladder mucosa invariably presented the structure in its early form, except in that of Pendl, where no active areas were discovered, and in that of Barringer, where the tumour did not penetrate the mucosa.

The macroscopical and microscopical aspect as seen in the region of the bladder will accordingly be considered next. The former depends to some extent on the anatomical structure of the urachus at this level in the particular individual concerned.

Cystoscopic Appearances.—

1. *Ulceration* (5 cases).—There was a very definite circumscribed ulceration, or rather appearance of ulceration, involving an area of 1 to 2 cm. in diameter (*see Fig. 324*). This area presented points of necrosis, and had a margin raised above the surrounding mucosa, which appeared to remain normal right up to the circumference of the ulcer. The latter was in some cases slightly excentric towards the right or left.

2. *Papillomatous Appearance* (4 cases).—The tumour appeared as a papillomatous area well defined and surrounded by normal mucosa. In one instance there was a grey granular circumscribed patch on the summit of an elevation which the pressure of the tumour had caused. In the cases of Lavenant and Michin there was a cone-shaped retraction at the apex of the bladder from which fronds of tumour emerged. These were evidently patients in whom the urachus had originally a trumpet-shaped dilatation at the point of communication with the bladder (*see Fig. 336 A*). In Green's case there was a small opening 3 mm. in diameter through which gelatinous material emerged.

3. *Bulging of the Bladder Wall*.—In 3 cases the mucosa was intact, or only corrugated through adhesion to the growth.

In the remainder the cystoscopic appearances are not clearly described.

Microscopic Characteristics.—Alike where the bladder defect is flat and where polypoid or papillomatous protrusions are present, the surface is covered with a layer of high cylindrical epithelium only interrupted at the points where necrosis has taken place or at the tips of the papillae. In de Korte's case the surrounding transitional epithelium was becoming transformed into the columnar type. On this surface, according to Albarran, open the ducts of the glands which lie in the wall of the bladder. These take on complex and branched forms, and the acini lie directly among the muscle fibres. They are surrounded by young fibrous tissue which tends to replace the muscular elements and form cores for the papillae which project into the bladder.

The cells lining the acini are of the high cylindrical variety with basal nuclei. They exhibit the various stages of the formation of mucin. In some it exists in the form of small granules, in others in droplets, and in others again these droplets have run together to form a central mass. In some places the defined free margin of the cell has ruptured and delivered its mucin into the acini. The secretion of mucin is great, and the quantity delivered in the urine very noticeable. Cells of the same type push into the fibrous framework, and are seen multiplying by an amitotic process. Large typically carcinomatous groups of cells may also occur in the stroma.

In many places there is more than a single row of cells, clearly apparent in my specimen, and described by most of the authors who have given attention to the microscopical detail. Instead of the cylindrical epithelium resting on the underlying stroma, it is based on another row or two rows of cells. The latter are cubical and flattened, but their axis is irregular. *The impression is given that the topmost row of a primitive transitional epithelium such as exists normally in the urachus has taken on the rôle of secreting columnar epithelium.* This is a most important indication of the real origin of the tumours. There is no membrana propria, the cells being in direct contact with the fibrous tissue of the stroma. The epithelium of the urachus has likewise no membrana propria, and its absence in the case of doubtful neoplasms is of considerable value in assigning the correct etiology. (*See Figs. 334, 344*).

Appearances in the Older Parts of the Growth.—The structures rapidly degenerate as one passes upwards from the bladder mucosa. Masses of mucin are seen lying free in spaces formed by fibrous tissue, and even apparently

among the individual fibres. In these masses may be seen 'ghosts' of degenerated cells, fragments of nuclei that still take the stain, degenerated red blood-corpuscles, and sometimes remains of fibrous cells. In some of the spaces, more especially the smaller ones, the process of degeneration is not so advanced. Rows of cylindrical cells adhering to each other line small sections of the wall of the cavity, or are tilted up from this at one end by the extravasation of blood between them and the lining membrane. Pale nuclei may still be seen here and there at the base. In other cases the epithelium has become flattened from pressure, and all grades are seen from low cylindrical, through cubical, to cells as flat as pavement cells. The cubical cells seem in some cases to represent the highest development of the urachal epithelium. *In the cases of Barringer and Pendl they were actively secreting.*

In some instances, and especially in the larger cavities, there is more than one layer of cells lining fragments of the wall. Ruptured alveoli, and alveoli lying back to back without intervening fibrous tissue, may also be seen. The whole field is saturated in mucin, not only in the cavities but between the fibrous bundles of the stroma. Large cells with vesicular nuclei may be seen penetrating the fibrous tissue. There are no formed blood-vessels except in the stroma, but a good deal of free blood in some of the larger cavities, giving a red jelly-like appearance to the content. Yellow streaks rich in cell membranes and debris can sometimes be distinguished from the surrounding white masses.

In Pendl's case the cells were all of the cubical variety. He likened the appearances seen to those in the older parts of a colloid cancer of the rectum. In my sections there were certain areas in which the mucus-secreting elements did not predominate, but in which the cell arrangement was that of the young transitional epithelium, typical of that in the urachal canal.

Some Remarks on the Nature of these Colloid Cancers.—While the general appearances of the tumours bear a strong resemblance to rectal cancers of a similar type, there has been abundant opportunity to prove by operative examination and autopsy that they are primary, and indeed a very close investigation will reveal certain points of difference from intestinal growths and betray the true origin of the apical neoplasms. Some of these features have already been indicated.

"Rectal cancers as a rule produce a more developed and permanent type of granular cell which holds its form longer and is more stable than that in urachal tumours" (de Korte). In the latter, so great is the production of mucus, and so rapid the breakdown of the cell, that the formed elements occur over a very limited area. The cell goes through its furious cycle of mucin production and is soon destroyed by its own activity. So copious a production of mucin also catches, unawares as it were, many cells before they can undergo the process of metaplasia. The pressure of the enormous flood which finds no outlet 'fixes' on the walls of some of the cysts many rows of cells still in their primitive cubical or flattened form. They are crushed out of existence before they can take part in the fantastic riot and vicious activity of their fellows. Hence cubical or flattened epithelium still maintaining its primitive transitional formation is visible for a short period before it too becomes disintegrated in the general destruction.

Again, the metaplastic impulse is so violent that the topmost layers of cells become cylindrical and secrete mucus before going through the formality of arranging themselves in single file like well-ordered mucus-secreting cells. They exercise to the top of their bent their potencies, long held in reserve in the cellular lumber-room of the urachal lumen, while still in position as the topmost layer of the transition epithelium, and seem to endeavour by their frenzied activity to make up in a few days for the enforced inactivity of years.

The complete absence of a membrana propria has already been mentioned. De Korte notes the great thickness of the walls of the cells, as if they had undergone a process of hyperplasia.

CLINICAL HISTORY, DIAGNOSIS, TREATMENT, AND PROGNOSIS.

Before passing on to the rather more interesting subject of the etiology of the colloid urachal cancers, it is necessary to devote some time to the more practical aspects, in order to leave the field clear for the detailed study of the urachal cells and the transitional tumours, simple and malignant, which form stages on the way to the fully developed colloid cancer, or, falling short of this goal, branch into the less regular types, classified in this paper as 'mixed tumours'.

Clinical History and Symptomatology.—The clinical history differs from that encountered in vesical neoplasms in that certain symptoms due to the growth of the tumour usually occur before bladder trouble proper ensues; and indeed in one or two instances the growth was removed before it had actually penetrated the mucosa, and there was no disturbance of micturition. The exceptions to this rule are the small group which are malignant from the beginning, and penetrate into the bladder before they have had time to extend upwards. A number of tumours about the region of the vesical apex have been found at post-mortem examination. These had not caused the patients any trouble during life, and it can be well imagined that a growth must attain considerable size in the free space above the bladder before it gives rise to any symptoms at all. Occasionally by interference with the action of the urachus, which plays a by no means negligible rôle in bladder dynamics, there is some hesitation in starting the stream, mild frequency, and other bladder dysfunction—mere annoyances not sufficient in themselves to take the patient to his doctor. In Pendl's case, for example, in which an enormous tumour the size of an adult head was found, with a capsule in places 2 cm. thick, and composed of old sclerosed and avascular fibrous tissue with areas of calcareous deposit, the patient made no complaint until two months before examination, although the growth had probably existed for years.

In Green's case there was some frequency of micturition *fourteen years before*, which then subsided, and the patient was conscious of little disturbance until he himself felt the orange-sized tumour one year before he was examined.

The evidence seems to prove that at first the growth is non-malignant and produces no symptoms until it becomes very large, unless it breaks through into the bladder.

The first complaint, as a rule, is abdominal discomfort, sometimes itching, and tenderness when the patient presses his own abdomen—and at times

discovers the presence of a lump in the process. Often the first symptom is hæmaturia, and this, in most cases though not all, is followed by the inflammatory phenomena of pain on micturition and marked frequency. Later, pieces of tumour may be found in the urine. The inflammatory phenomena may mask the slight hæmaturia, as, in common with all other cases of malignant tumour involving the bladder, infection is apt to set in early.

On examination an abdominal swelling may be seen or felt, movable in its upper part, fixed in its lower. Dullness on percussion may be elicited, and, more commonly, even where no tumour is palpable, there is tenderness on deep pressure in the suprapubic region. Bimanual examination by rectum or vagina may elicit a tumour and locate its site as being high up in the bladder, or there may be intense tenderness during the examination.

Cystoscopic examination, to be fruitful, requires that the examiner be aware of the existence of such growths. They may be present merely as swellings or protrusions of the bladder apex, giving the impression that something is pressing from the outside. There may be merely what appears to be a flat epithelioma, though the circular form and the presence of normal bladder mucosa up to the very circumference of the lesion are marked features. In these cases there is usually a raised edge, and a small piece removed for examination will show the typical structure. In others there may be a funnel-shaped retraction of the bladder apex with a tumour ring around it or a tongue of growth emerging from its recesses. In still others the appearance may be of a localized tuft of polypi or papillomatous fronds projecting into the bladder. In Green's case there was a small hole such as may be seen in cases of normal urachus, but gelatinous fluid was being discharged from it. Wherever possible a small piece should be removed for examination so that the error of treatment by fulguration or by radium may be avoided.

Differential Diagnosis.—The growth is to be distinguished from the common types of tumour found in other parts of the bladder wall. The rarity of these at this site, however, demands a thorough clinical examination for the presence of a suprapubic tumour, and the removal of tissue for study. If the tumour is palpable above the symphysis, the differential diagnosis from neoplasms of the omphalo-mesenteric duct, echinococcal cysts, abscesses in the space of Retzius of urachal or other origin, desmoid tumours, lipomata, and cysts arising within the abdominal cavity, has to be considered. This presents little difficulty if the possibility of the occurrence of the urachal tumour is kept in mind. Early adenomatous formations arising from the urachus must be distinguished from endometriomata, whose structure they may closely simulate.

Age Incidence (by decades).—

YEARS		CASES
20-30 1
30-40 2
40-50 7
50-60 3
60-70 4
70-80 0
80-90 1
Total	..	18

No case younger than 29 has been recorded, and the larger number of the patients were over 40. Examination of the urachus from routine specimens shows that the small adenomatous formations do not develop to any extent until middle age. In this respect there is a correspondence to Lendorf's observation that the glandular structures in the bladder develop very slightly till after puberty, and are much more frequent and widespread after middle life.

Sex Incidence.—Of the 18 cases, 14 occurred in males and only 4 in females, showing a decided preference for the male sex. The significance of this feature is not clear, but it is difficult to associate with the richer development of prostatic glandular tissue in the male.

Treatment.—The treatment is based on the knowledge that metastases are rare, but that since they are very common after interference the operation must be radical, and based on the consideration that one is dealing with a tumour in the urachus whose connections, direct and indirect, may reach in abnormal cases as far as the umbilicus. Metastases after operation occur not uncommonly in the space of Retzius. The tissue removed must include the umbilicus with an area of at least half an inch of skin round this, a wedge-shaped block including the transversalis fascia and peritoneum wide of the growth, and the upper part of the bladder. In deciding on the amount of bladder to be removed, consideration should not be given merely to the area of mucosal involvement, because the part of the muscular wall involved is always greater in diameter (*see Fig. 325*). At least the upper half of the bladder should be removed. In the past there has been an attempt, always doomed to failure, to preserve the peritoneum. The opposite principle should be adopted, as much of this structure as feasible from the point of view of securing apposition being removed with the growth.

Prognosis.—No evidence is available as to the malignancy of these tumours if left alone. As they resemble colloid cancer of the bowel in the fact of the mucin being secreted by the cells, rather than the relatively benign cancers of the breast* where it may be in part due to degeneration of the stroma, one would expect them to be very malignant.

The long history in most cases indicates rather that the cancer has superimposed itself on a non-malignant tumour than that it is in itself of a relatively benign type. The evidence on this question, however, is incomplete. There is more information available in regard to the progress after operation. My case died two and a half years later from metastases without bladder recurrence. No report earlier than this can be safely considered as an index of cure.

Not one of the 18 cases of the series which was followed up for more than two and a half years was alive at the end of that time with the single exception of Lavenant's patient, who was apparently well but had had a hemiplegic attack, the latter being in itself significant from the point of view of metastases. On the other hand, the immediate operative results were good, and in

* My acceptance of the traditional view that colloid cancers of the breast are relatively benign has been modified since writing the above after a conversation with Dr. Broders of the Mayo Clinic. In the abundant material at his disposal Dr. Broders has found that 90 per cent of such cancers must be graded as 3 on his scale, and that in the formation of metastases and resistance to treatment they are on the whole more malignant than non-colloid carcinoma.

many cases there had been no recurrence in the bladder after one year. Metastases elsewhere do not as a rule declare themselves in that time. This seems to show that the growths are of only average malignancy.

Secondary neoplasms of the bladder derived from other organs through the blood-stream are very rare; secondary colloid cancers of the type under discussion have not as yet been recorded. A few clinical cases are reported in which metastases of the bladder apical tumour were widespread. In these the growth was found at autopsy. De Korte's case probably had a large mucinous mass in the pelvis before operation, but in none of the others was there any reason to believe that metastases were present before operation. They occurred afterwards in the abdominal wall and the bladder itself, but it must be remembered that the operations planned did not conform to the radical one I have suggested.

A SURVEY OF THE REMAINING CLASSES OF ADENOMATOUS AND 'MIXED TUMOURS ARISING FROM THE URACHUS AND INTIMATELY INVOLVING THE BLADDER.

The clinical study of the colloid adenocarcinomata of the bladder vault has now been completed. Before proceeding to an examination of that chain of events by which the primitive cloacal cells of the foetus become the active malignant ones of the adult, it will be as well to consider shortly in some of their clinical and histological details the two groups which have been referred to as the adenocarcinomata and the 'mixed tumours' (*see* p. 434). Following that, a brief reference will be advisable to the group of mesodermal growths, which in my opinion arise from extra-urachal structures. These have, of course, no bearing on the etiology of colloid cancers, but as they have been described again and again as tumours of the urachus, they cannot be altogether ignored in a study concerned with that structure. There remains the group of urachal rests (*Class IV*, p. 434). Finally, a brief reference will be made to the adenomata of the umbilicus, in order to illustrate the similarity in form which growths derived from the foetal hind-gut assume when they take on neoplastic activity in the adult.

Adenocarcinomata without Colloid Degeneration.—It is a striking fact that so few examples of simple or non-colloid adenocarcinoma have been reported—only three in all (*see* (d) and (e) in table on p. 434).

It is remarkable that while in the case of the colloid growths there is reason to believe that the results were uniformly bad, two of the three cases of adenocarcinoma were well after nine and ten years respectively, one of them after having had 12 in. of intestine removed with the tumour.

In some instances of colloid formation—illustrated, for example, in the reports of Barringer and Pendl—the cells had taken on active secretory activity while still cubical. Taking this into relationship with the paucity of cases of simple adenocarcinoma, one is led to expect that the phase of columnar-celled formation is practically synchronous with mucin secretion and malignancy. The urachal cells hasten feverishly to begin the activity which as entodermal cells has long been denied them, not even waiting to assume the regulation cylindrical shape before starting their secretory riot. In the three

cases of adenocarcinoma, where they had become frankly columnar and still exhibited but little energy, we are doubtless faced with an exceptional instance of unusual tameness and docility on the part of the epithelium.

'Mixed Tumours'.—The term 'mixed tumour' in this connection is somewhat of a *petitio principii*, as it is doubtful if the neoplasms so described were really of the nature of teratomata. The group, however, present unusual and diverse pathological features, and more than one author considered that his case was an example of a true teratoid tumour. Ewing classified Barringer's specimen as such, though it seems to fall in line with the other colloid adenocarcinomata. The reports of the cases here referred to (*see* (g) and (h) in the table on p. 434) all concern neoplasms closely connected with the bladder apex. They were considered by those who reported them to be derived from urachal or 'allantois' remnants. Some of them present features which remind one very forcibly of certain formations found in connection with the urachus, and it is possible that they represent an unusual malignant development of the microscopic adenomata. Similar types are not unknown arising from the bladder mucosa proper (*see* p. 437).

Some of the tumours which contain sarcomatous and epithelial elements side by side are probably examples of growths in which both the mesodermal sheath and the epithelial canal take part. In some cases the derivatives of the former predominate, giving a picture of a sarcoma with a few glandular spaces. The competition of the mesodermal section modifies the form of the acini, but the cubical cells, sometimes of more than one layer, which line the spaces and the papillary projections are reminiscent of certain urachal appearances.

Mesodermal Growths Arising from the Muscular and Fibrous Tissue of the Urachus and from the Ligaments connecting the Urachus with the Umbilicus.—All the ten cases (*see* (m) and (n) in the table on p. 434) were of the same type. They did not arise near the bladder, and it is probable that all developed from the strands or ligaments connecting the urachus with the umbilicus. They have no bearing on the subject under discussion, except that they are described and reported as urachal tumours. Most of them occurred in females, in contradistinction to the true urachal tumour, which as a rule is found in adult males.

Reference has now been made to all reported bladder apex tumours which appeared to originate in the muscular wall, as far as I have been able to find them in the literature. In addition, all growths which have been thought by the authors to be of urachal origin have been commented on, with the exception of the cases of Nicholson, Hector, and Hagner, which were simple fibro-adenomata discovered at autopsy or exploratory operation and will be discussed later, and those of Koslowski, Adam, and Hue and Jaequin, which are successive stages of neoplasms developed from urachal rests above the adult position of the urachus itself. These also will be considered in a later section.

There has been no selection or elimination of cases to prove a thesis. The striking fact emerges that the thirty-one tumours arising from the bladder apex were either colloid adenocarcinomata, adenocarcinomata, fibro-adenomata,

or 'mixed tumours' mostly having a very definite glandular aspect. The cases of Doiran and Cullen cannot be definitely classified, but they were unusual growths of uncertain nature. On the other hand, the ten tumours which arose from mesodermal tissue all developed above the bladder, and involved the latter, if at all, only by direct spread along the abdominal wall.

This is a striking confirmation of the anatomical findings that the urachus with its epithelial canal extends only 5 to 8 cm. from the bladder apex. It also seems to justify the statement that true tumours of the urachus always have an epithelial element, and that those which do not satisfy this criterion should be discarded from the category.

PART II.

THE ORIGIN AND HISTORY OF THE EPITHELIUM OF THE URACHUS, AND THE PATHOLOGICAL CHANGES IN IT WHICH LEAD TO THE DEVELOPMENT OF THE COLLOID ADENOCARCINOMATA.

The primitive hind-gut in the early foetus is the anlage of the rectum, the upper part of the bladder, and the urachus. This cavity is, according to Wesson,¹³⁰ lined by a single layer of cuboidal cells. A tendency for these cells to become more cylindrical in shape on the one hand, and to form by their subdivision two superimposed layers on the other, may be observed very early, adumbrating the tendencies which are finally brought to fruition in the formation respectively of the cylindrical mucosa of the rectum and the transitional epithelium of the bladder. The complete differentiation of the types takes place only after the interposition of the urorectal septum has determined the limits of the two cavities. Equal potencies are inherent in all the cloacal cells, and the ultimate form which they will assume is determined only by the physiological rôle assigned to them. The exact point of subdivision effected by the urorectal septum is arbitrary to this extent, that it does not separate a group of cells predestined to form rectal mucosa from another group equally predestined to form transitional epithelium, following as it were a trail already blazed where two histological continents march side by side, or building a wall along an international frontier previously determined. Such exactitude is at variance with our experience of embryological actualities. The further development of the individual cells is decided by the position which the septum takes up rather than the latter by any inherent difference in the nature of the cells.

While this view is probably so universal that its expression is almost a platitude, the theory of rests or abnormally persistent rectal epithelium has much acceptance, but appears to be entirely illogical. Misplaced intestinal mucosa has been invoked to explain colloid cancers occurring in the bladder area. The only assumption on which such a proposition can rest is that certain areas of the epithelium of the cloaca have limited potencies compelling them if they persist to become rectal mucosa in the case of certain cells on the one hand, or transitional epithelium in that of others—the two groups being fundamentally distinct. After the urorectal septum has formed, the cells of the former group which find themselves anterior to the partition are said

to die out, as they have no power to become transitional epithelium, and find themselves accordingly in an anomalous position. If they persist they remain as 'rests' of rectal mucosa and may give rise to appropriate types of tumour.

If this theory were correct, one would expect to find intestinal growths in the bladder as the rule rather than the exception. The proposed axiom of the death of cells which find themselves in a teleologically unsuitable situation is hardly less fantastic than the supposition that the urorectal septum will neatly divide the narrow line between two individual cells of an entirely different nature.

The whole theory of vestigial rests has been worn threadbare to explain difficult pathological phenomena. The only logical point of view is to think of primitive cells* such as exist in the early cloaca as being totipotent as regards the formation of neoplasms ectodermal, endodermal, or mesodermal. The assumption of the specificity of the three primary germinal layers belongs to an older pathology. The theory certainly had its attractions, for by juggling with this trinity of neoplastic ancestors and using the trump card of vestigial rests to retrieve a losing trick, a more or less plausible explanation could be given of any and every pathological phenomenon! When, however, we meet with squamous epithelioma of the gall-bladder, uterus, bladder, and kidney pelvis, carcinoma of the mesodermal renal tubules, and similar phenomena, our credulity in this axiomatic specificity is stretched to the breaking-point and inevitably compels a belief in a higher potency latent in body cells than the older pathological opinion would admit.

While multipotency, if not totipotency, must be admitted for all cells even up to an advanced period in their life-history, there remains a strong tendency for the three primary layers, ectoderm, mesoderm, and endoderm, to develop along their own special lines. Any idea, however, of their absolute specificity ought to be discarded, and metaplasia looked on as a phenomenon not excessively rare.

Returning to the primitive cloaca, we find that the cells posterior to the urorectal septum develop into the highly organized structures of the intestinal mucosa with its glands, its stately columnar epithelium, and its mucous cells; those anterior into the well-morticed architecture of vesical epithelium. Hardly have these baby cells of the hind-gut begun to develop, however, than a further subdivision takes place—a partitioning of a very different type from the former, which reproduced in ontogenetic miniature the phylogenetic emancipation of the higher genera from the disadvantages of the rather too universally applicable cloaca! This second subdivision, although humbler in its object, is not without purpose. By it the upper part of the bladder is narrowed down as a prelude to the descent of that viscus behind the safe shelter of the symphysis pubis. This migration has a double aim. It guarantees the organ

* The limit of age beyond which adult epithelium becomes incapable of metaplastic change to another type—if indeed any limit exists—is undetermined. That totipotency as a quality is not confined to primitive cells is evidenced by the formation of ectodermal neoplasms from developed endodermal varieties, as seen in the squamous-celled epitheliomata of the gall-bladder as well as the urinary bladder. There is probably a reversion to a more elementary type of epithelium as a prelude to the metaplasia (see p. 430 and *Fig. 339*).

against injury, to which its fixed position outside the peritoneum and well to the front of the abdominal wall would expose it, and at the same time removes its apex from the weak point of the umbilicus, rendering more secure this final point of closure of the foetal leaf, and removing the danger of urinary fistula involved in such intimate association between a frequently distended viscus and a vulnerable point in the abdominal wall.

It is not advisable, however, that the bladder should lose entirely as its *point d'appui* such a convenient point as the umbilicus, since the vesical apex must be steadied to fulfil satisfactorily the function of micturition. The formation of the musculo-tendinous apparatus of the urachus and the ligamentum commune fulfils all requirements (*see Fig. 328*).

The ideal is, however, not absolutely attained, for the epithelial canal left in the urachus has no possible place in the physiological scheme. The epithelia of the bowel and bladder fulfil their respective destinies to meet the needs of the organism; that of the urachus has no destiny at all. It is merely flotsam and jetsam, builder's refuse thrust out of the way among the joists. But though its *motif* is lost, its vital impulses remain. It does not die, and yet it has no useful purpose in living. It remains fixed at that stage of development which it had reached at its segregation, primitive epithelium. There is no imbrication, mortieing, and welding of the cells with each other—the mutual adaptation by which their more fortunate brethren co-operate to form the perfect waterproof lining of the bladder. The urachal cells remain individualists. They heap themselves up on each other without order or regularity (*see Figs. 334, 335*). That the masses of embryonic elements form a complete lining to the urachal canal many layers in depth is an accident of reproductive activity rather than the end-result of any teleological aim. These pariahs and outcasts from the corporal community exhibit the unbridled procreative powers of the unfit. Primitive cell multiplies from primitive cell in the narrow prison house. Reproduction and degeneration, degeneration and reproduction, follow one another in unending eyele. The cells jostle and destroy their fellows in the struggle for room in which to exist. Individuals perish, and the débris from their remains fills the narrow urachal canal from which there is no escape. The force exhibited in cellular reproduction is immense. By its means the tender weed forces its way through solid asphalt, and roots wedge rocks apart. In the urachus the new cells formed at the periphery, finding no space in the crowded lumen, press out into the sheath; and in that narrow and unguarded area where the tube, devoid of any fibrous protection, passes through the muscular bladder wall to reach the mucosa, the expansion and contraction incidental to the act of micturition alternately release and compress the cellular column and give still further impetus to the unbridled behaviour of its inmates. In this way the scene is set for the formation of these irregular cell-growths which predominate in later life, but which fortunately progress as a rule too slowly to become a menace before death ensues from other causes.

The retention by the urachal cells of all the potentialities of primitive cloacal cells, and their ability to bring them at any period of life to untimely fruition, are the keys to the understanding of the various types of tumour of the urachus. Complete but ill-regulated development towards glandular

or transitional epithelium respectively may be observed, may overstep all bounds, and give rise to colloid adenocarcinoma on the one hand or squamous-celled epithelioma on the other. Both these types were seen in a single tumour in the sixth case of Rankin and Parker. As a rule, however, the full impulse given by the entodermal origin of the cells predominates, and the formations commonly seen pass through the stages of simple adenoma and adenocarcinoma to reach finally the fully formed colloid cancer of the rectal type. It is fortunate that this transformation starts as a rule too late or progresses too slowly to reach the threshold of clinical observation during the normal span of life, but it may commonly be observed by microscopic study of the urachus in routine autopsy specimens. Sufficient points of resemblance exist between these microscopic tumours and those of clinical importance to enable them to be identified as different stages of the same process.

RELATION OF URACHAL ADENOMATA, ETC., TO THOSE ARISING FROM THE PROPER EPITHELIUM OF THE BLADDER.

What has been stated in regard to urachal epithelium applies also in a modified sense to that of the bladder itself. The cells of the latter are of the same origin and theoretically retain the same latent potencies. In point of fact, adenomata, adenocarcinomata, and colloid tumours of the rectal type *do* occur in the bladder, and have been proved to arise from the epithelium of that organ. The transitional epithelium may become converted into cubical, the latter into cylindrical, and finally adenocarcinomata of the intestinal type, with or without the excessive secretion of mucin, may result (*see Fig. 339*). The primitive urachal cells, however, which retain their early cloacal characteristics, may develop along the divergent paths which lead towards vesical or rectal epithelium with equal facility. On the other hand, those of the bladder have already advanced a considerable distance along one of these diverging paths, and to be converted into the rectal type must either retrace their path to the starting-point and then proceed by this circuitous route to the goal of intestinal mucosa, or, alternatively, make the incredibly difficult metaplastic leap over the gulf which separates the transitional from the rectal type. Considering the enormously greater quantity of epithelium in the bladder compared with that in the urachus, these changes are correspondingly much rarer in the former organ than in the latter.

MICROSCOPIC ADENOMATA AND CYSTS OF THE URACHUS, AND THEIR FORMATION.

Anatomical Considerations.—We know that the urachal canal is surrounded by a stout fibromuscular sheath as far as where it pierces the superior bladder wall, and that below this point it passes to the mucosa without any such protection, lying free in the muscular coat of the bladder. In conformity with this anatomical fact it has always been found that malignant tumours are fairly well encapsulated above, but invade the tissues unhindered in the intramural portion.

If a number of specimens of the urachus are taken at random, it will be found that the canal can be easily dissected out from its sheath, which hides

the irregularities in its contour; and it will then be found in a large percentage of cases to exhibit nodulations and tiny cystic formations, more especially in those parts that lie immediately above the bladder and actually in the vesical wall (*see Fig. 337*). Where the lumen fails to maintain connection with the bladder cavity, the blind end at the level of the external surface of the bladder mucosa is frequently dilated to form a bulbous swelling microscopically similar to the cysts projecting from the lateral wall (*see Fig. 338*).

Luschka⁶⁰ observed diverticula and cystic formations in the urachal canal in most of the specimens examined. In the 74 cases of Wutz¹³² no fewer than 24 exhibited cystic dilatations. In a majority of the ones I have examined



FIG. 340.—Section taken through the muscular wall of the bladder apex. There are two urachal canals. One has been cut partially in longitudinal section.

there were either definite projections as large as barley grains visible to the naked eye, or microscopical examination revealed multiple canals parallel to the main one (*Figs. 340, 348*), columns of cells lying free (*see Fig. 346*), small cysts lined by cubical or flattened epithelium (*see Fig. 343*), or complex structures having the appearance of fibro-adenomata (*see Fig. 344*). The cysts appear in all cases to be derived from degeneration of the central portion of solid columns or masses of cells in the first instance, with successive multiplication of the limiting cells and degeneration of the more internal ones, until comparatively large cavities were formed (*see Figs 341, 342*). It seemed very questionable if any of the contents of these *early* cysts owed their origin to

actual secretion from the lining cells. They were true cystic adenomata, and not due to retention of secretion.

These microscopic structures have probably no relation at all to the enormous cysts appearing in the literature under the caption of urachal cysts. The latter owe their origin to pathological processes of various kinds, mostly having little or no connection with the urachus. The small adenomata and cysts found on examination of routine specimens have, however, their counterpart in larger structures, such as the specimens reported on by Nicholson⁸⁷ and Hagner.⁴⁹ A more advanced stage may be seen in a specimen of a spindle-shaped cyst $2\frac{1}{2} \times 1$ cm. in cross-section in the Museum of University College Hospital, London. It was found incidentally during an operation on a woman for some pelvic condition.

Changes in the Clinically Normal Urachus.—Some examples of the various changes to be found in the clinically normal urachus will now be described in detail and illustrated by specimens selected to demonstrate the different phases, beginning with the earliest and passing on to the more complex.

The following is in general the sequence: The cells of the urachal canal are constantly proliferating, by the amitotic method. The effect of this is twofold. On the one hand, they become heaped up towards the lumen, some being desquamated into it (*see Fig. 334*); on the other, the peripheral cells extend in a centrifugal direction and are cut off from the main column by the ingrowth of the fibrous tissue of the sheath (*see Fig. 341*). The separated packets continue to multiply, forming masses, but more generally extending as solid columns for some distance parallel to the direction of the urachal canal. By the degeneration of the middle cells, presumably from insufficient blood-supply, a lumen is formed. A cross-section at this stage will exhibit the large central urachal canal flanked by other smaller ones, all lined by several layers of cells (*see Fig. 340, 346*). When the separated groups assume the form of a mass rather than a column, the extension by multiplication of the peripheral units, together with the successive degeneration of those situated towards the centre, leads to the formation of a cyst filled with amorphous content and the remains of cell membranes and nuclei (*see Figs. 345, 347*). The increase in circumference of such a cyst produces a thinning out of the epithelial lining, and the pressure of the contents a flattening of the lining cells. There may be only one or two layers of the latter.

If numerous masses and columns of cells are cut off rapidly, the neoplastic appearance of an adenoma is formed in which both solid columns and cystic structures appear side by side, and the cells in such cases may change from their primitive form and become elongated, cylindrical, and finally actively secreting cells. At this point the tumour has definite malignant tendencies.

Another feature observed is where the fibrous stroma pushes into a cystic cavity in a tongue-shaped manner carrying the epithelial layers with it and forming a papillary projection (*see Fig. 350*). The fibrous core of such a papilla is covered with the layers of the epithelium originally lining the walls of the cyst which it has pushed in front of it when it invaded the cavity. These papillary formations are a prominent feature of some of the 'mixed tumours'.

Specimen 1.—Illustrating the Formation of a Urachal Cyst (*Figs. 337, 341, 342*).—The urachus was taken from the body of an old man undergoing dissection. No clinical history was available. The specimen had the appearance of a normal urachus and was 5 cm. in length. The canal extended to a point 4 cm. above the bladder. On removing the fibrous sheath two small smooth cystic projections

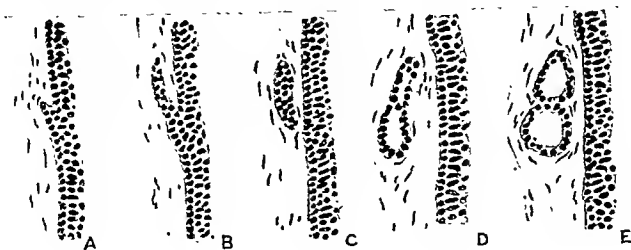


FIG. 341.—The beginnings of a cyst. Sections taken from above down in specimen shown in *Fig. 337*. A, Epithelial bud into the fibrous tissue; B, A mass of cells formed still attached; C, The mass is separated from the parent epithelium; D, The central cells have degenerated and subdivision is commencing; E, Subdivision is complete.

were seen projecting laterally from the outer wall of the structure at a point 1 cm. above the bladder mucosa (*see Fig. 337*). They were sessile, 2 mm. in diameter, and their thin capsule was formed by an expansion of the fibrous coat of the canal.

MICROSCOPICAL EXAMINATION.—Serial sections were cut from the upper level of the urachus to the bladder, including the cysts.

The canal was of normal appearance till a point was reached 1 mm. above the level of the upper cyst (*see Fig. 337*). Here the outermost cells of the epithelial lining formed a little group of three or four budding into the connective tissue (*Fig. 341 A and B*). A little lower the group of cells was larger, and was separated from the canal by young fibrous tissue (*Fig. 341 C*). Still lower the central individuals were breaking down (*Fig. 341 D*), thus forming a lumen surrounded by several layers of cells. The small cyst thus formed was continuous with that microscopically visible below. It had divided into two, but the cavities as seen in the section had again connected with each other and with the lumen of the urachal canal (*Figs. 341 E and 342*). An accumulation of cells marks the point of amalgamation of the two epithelia, that of the cyst and that of the urachal lumen. Both lumina contain amorphous content which has shrunk away from the walls in the process of hardening.

The steps in the formation of this cyst may be summarized as follows:—

1. Bud of cells pushing out into the fibrous membrane lining the canal.
2. The multiplication of these cells, forming a mass separated by young fibrous tissue from the canal proper.
3. The breaking-down of the central cells forming a lumen, and the division of the hollow columns into two.

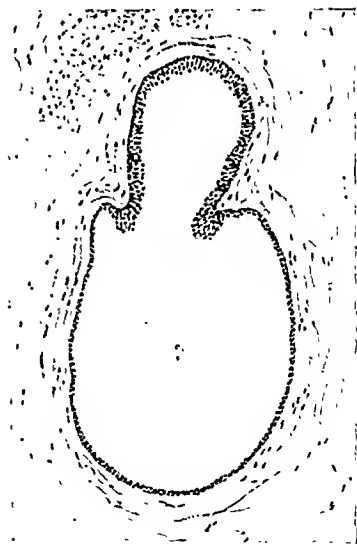


FIG. 342.—A section through the upper cyst shown in *Fig. 337*. The new cyst has again joined the urachal canal, which shows some dilatation. Note the thicker epithelium of the cyst, the amorphous substance filling the cavities, and the degenerated nuclei in the canal. The cyst content has shrunk in the process of hardening.

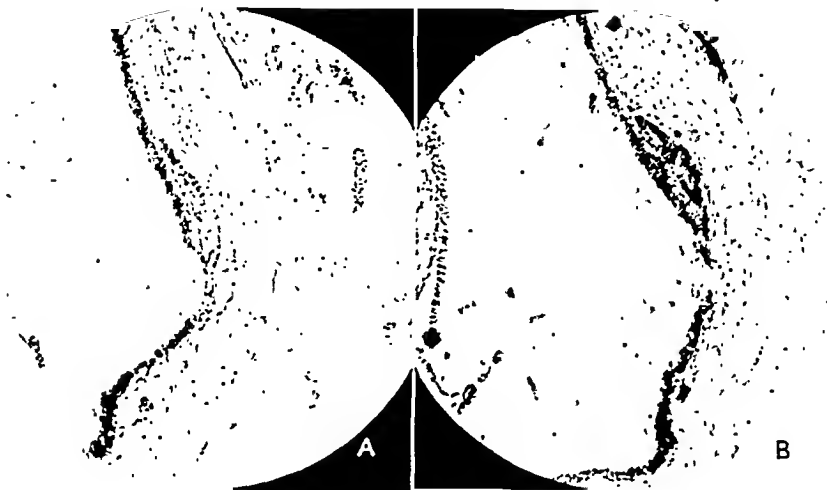


FIG. 343.—The upper and lower of a group of serial sections taken from a small cyst at the lower end of a urachus. In A a group of cells is isolating itself from the urachal epithelium. In B two small cysts have formed from this group lateral to the main cyst wall.

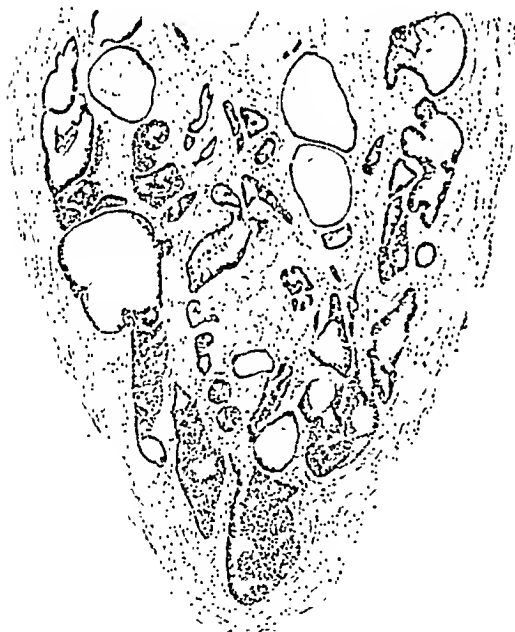


FIG. 344.—Section of the lower part of the supra-vesical urachus from a woman of 47 years. The illustration represents half the total area of the urachus. There is adenomatous formation, and the growth has invaded the fibrous sheath.

4. The gradual increase of these separate cysts till their cavities have again burst into each other and into the urachal canal.

Lower down, the urachal lumen and the cyst which had burst into it became one homogeneous whole, so that the section through the lower cyst shown in Fig. 337 presented the appearance of a wide dilatation of the urachal canal. The lining epithelium consisted of only two layers of very flattened cells.

The tendency to subdivision either directly or after the lumen has again filled up with cells is illustrated in Figs. 342 and 343. The specimens were from two separate cases.

Specimen 2.—Illustrating the Formation of an Adenoma of the Urachus (Figs. 344–348).—The specimen was removed from the body of a woman of 47 who had died following the operation of hysterectomy. The urachus was 6 cm. in length and had a normal appearance. A section made just above the bladder illustrates a latent type of adenomatous formation (Fig. 344).

FIG. 345.—A series of high-power views of the same section as that shown in *Fig. 344*, showing how a round cavity lined by flat single-layered epithelium is formed by the degeneration of the central part of a column of cells. The debris resulting from this process filled the lumen, but retracted during the hardening of the tissue.

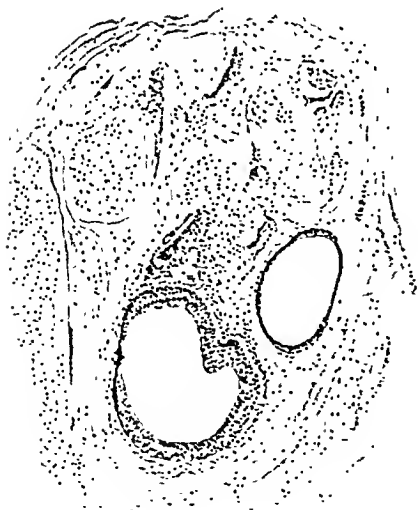
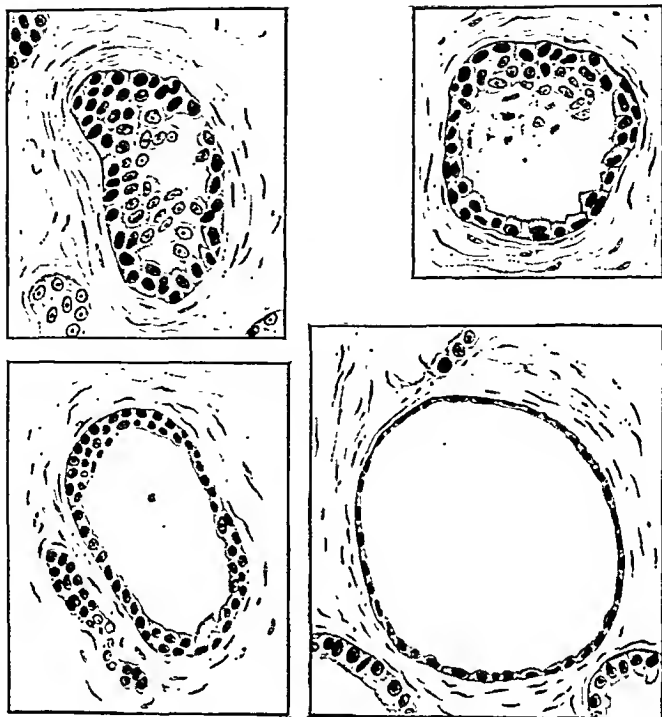


FIG. 346.—Section taken from the upper part of the same urachus as shown in *Fig. 345*. Note the two canals, the difference in the number of layers of epithelium, and the mass of detached cells above the canal on the left.

MICROSCOPICAL EXAMINATION.—Serial sections from the upper end of the urachus to the bladder illustrated the formation of the adenomatous structure (Figs. 346, 347). Above, the adventitial and muscular layers of the urachal sheath were normal. The lumen was at first single, but soon divided into two separate canals, each lined by the customary primitive transitional epithelium (Fig. 346). At the upper pole of one of these canals a group of cells could be seen pushing out into the fibrous membrane outside the epithelium, and forming a solid mass or column. In a lower section this mass had broken down in the centre and formed a lumen. The new canal became separated from the old, so that in the next section there were three canals all lined by the same type of epithelium. As the sections proceeded downwards, more and more of these groups of cells became separated, formed lumina, and continued as additional canals (Figs. 348 A, B, C). The process became widespread. Masses of cells detaching themselves came into contact with others already existing, and the figures became confused by the junction of two epithelial masses,

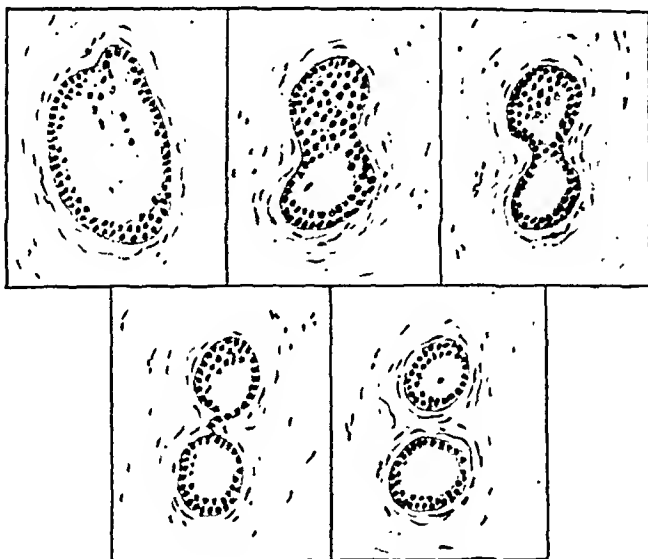


FIG. 347.—From the same case as that shown in Fig. 346. Thick serial sections from above down to illustrate the method of formation of two acini from one. Note the condensation of the fibrous tissue and the way in which it conforms to the subdivision of the acinus.

the amalgamation of two cysts to form one cavity, or the subdivision of one to form two. The section now began to have the appearance of an adenomatous tumour, and spread outside the fibrous membrane of the urachal canal proper, invading the muscular and fibrous sheath (Fig. 344). Towards the bladder below the sheath the acini, if they can be so called, maintained an orderly arrangement, did not penetrate between the muscle fibres, and stopped short at the external aspect of the mucous membrane—the lower limit of the urachus in this case.

Fig. 344 shows the structure at the widest point. Its boundaries are coterminous with those of the urachus. The adenoma has grown at the expense of the muscular sheath without expanding it. The outer adventitial coat has been thinned out, but still exists as a fairly regular fibrous capsule indented in places by the pressure of cysts nearest to it. The stroma consists of young fibrous tissue. All formations are seen, from irregular masses of epithelial cells having the general character of urachal cells, to cystic spaces. Most of the latter are surrounded by a condensation of the fibrous tissue. Numbers of them are scattered through the

stroma. The cavities contain an amorphous content which does not give the reaction of colloid (*Figs. 344, 345*).

The epithelial lining of the cysts varies. In some, apparently the more recent, the many layers of cubical epithelium similar to normal urachal epithelium are seen, while in other parts of the same cyst there is a single layer of cubical or flattened

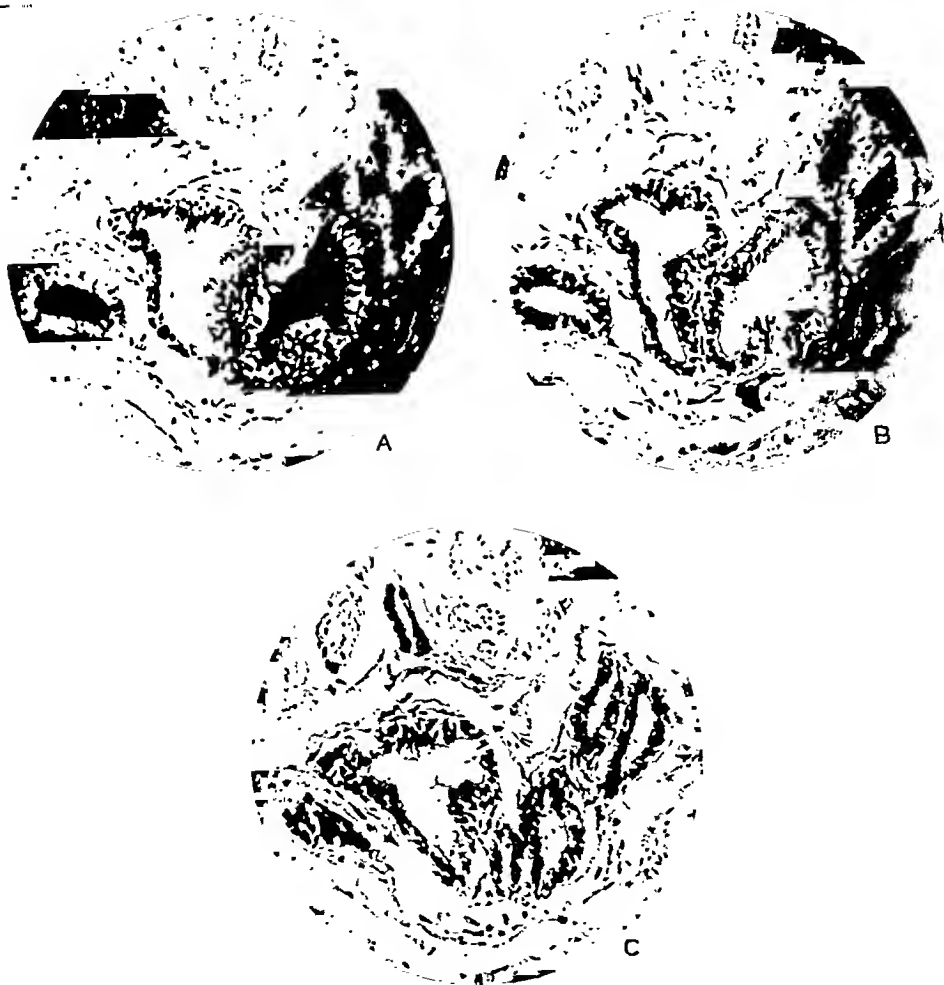


FIG. 348.—Three sections of the same urachus as shown in *Figs. 344–347* taken some distance apart to show multiplication of the number of canals, a process which finally produces the type of structure seen in *Fig. 344*. Two single canals in A have subdivided completely in C.

cells. In a few of the spaces where the cyst is not distended but has an acinar appearance the cells are becoming cylindrical, but there are no goblet forms.

A few of the larger cavities have a clover-leaf appearance with more layers of epithelium at the angles where the components join, suggesting that these have been formed by the amalgamation of two or more smaller cysts.

From the original urachal cells have been derived all the various types of epithelium seen—primitive, transitional, flat, all grades of cuboidal, and finally cylindrical. Similar varieties may be found in the developed adenocarcinomata. None of the cells have as yet started to secrete mucin, and there is no evidence of malignancy.

These specimens have been selected to illustrate the formation of a small cyst and adenoma respectively. The processes involved are essentially similar, and in the numerous cases where I have observed them exactly the same series of changes can be followed. The two selected are so typical, however, that nothing would be gained by multiplying examples. The process is always the same budding of epithelium, separation from the main canal, lumen formation through degeneration of the central cells, subdivision of cysts so produced, and finally the formation of spaces lined by cuboidal or flattened epithelium. In the later stages the tendency is for the cells to develop into cylindrical forms.

Goblet and mucous varieties were not seen in any of the specimens I examined, but Suehannek¹¹⁸ reports them in two of his cases.

FIBRO-ADENOMATA OF THE URACHUS AT THE BLADDER END.

This group follows naturally, as the cases represent a more advanced stage of the formations that have just been described.

NICHOLSON'S case.⁸⁷—

Male, age 47. The patient died of heart disease, and had had no symptoms referable to the growth. The latter was in the lower end of the urachus immediately above the apex of the bladder. It was 5.7 cm. in length from above downwards and 4 cm. in breadth antero-posteriorly. Thanks to the courtesy of Dr. Nicholson, I had the opportunity of examining the slides. The tumour had a fibrous capsule, and was occupied by a number of cysts and tubular spaces. The latter were lined by irregular columnar or cubical epithelium. Papillary projections from the stroma were seen in the glandular spaces. The author's illustration shows that these papillae are covered with cells tending to become cylindrical in type. In certain parts there is more than one layer lining the cavities, which contain amorphous debris similar to that found in the smaller specimens already described.

HAGNER'S case.⁴⁹—

Male, age 68. During an exploratory cystotomy a tumour was palpated projecting into the upper part of the bladder. It was submucous, and was attached to the apical region of the bladder. The growth was removed and sectioned.

EXAMINATION OF SPECIMEN.—The tissue consisted of glandular spaces in a fibrous stroma. There were 'inflammatory' processes in which the cells were degenerated.

Hagner gives no exact microscopical description of his specimen, but it appears to have been a fibro-adenoma of the same type as in Nicholson's case. The cells, however, were beginning to invade the stroma, and the tumour probably represented a transition stage between the simple fibro-adenomata and the adenocarcinomata.

Some years ago Dr. Hector, of Wellington, showed me a specimen of a bladder removed from the body of a man found dead. There was a spindle-shaped swelling the size of a walnut at the lower end of the urachus. The tumour was smooth and contained in an expansion of the urachal muscular sheath. Its lower end rested on the bladder. Microscopically it had an adenomatous appearance, but unfortunately the specimen and slides were

not preserved, and no detailed description was made at the time. In macroscopic appearance it was remarkably similar to the specimen shown to me by Dr. Nicholson.

As pointed out above, the clinical history of the majority of the cases of colloid cancer suggests that the malignant changes supervene in a simple tumour which has already been present for a considerable time. We can trace the steps in a fairly systematic manner. First of all the microscopic adenomata, then the larger types as seen in Nicholson's case, the same type showing evidence of early malignancy as in Hagner's, the adenocarcinomata with the beginning of mucin secretion in the cells as described by Hartmann,⁵⁰ and, finally, the fully formed colloid tumours.

In the case of the small and very malignant growths confined to the intramural urachus (cases of Kielluthner, Albarran, de Korte, and that of the author), the intervening stages appear to be absent, the urachal cells becoming actively malignant from the first. The observations of Suchanek¹¹⁸ are particularly interesting in this connection. He made a study of a series of specimens of the urachus, and not only confirmed the frequent occurrence of cystic and adenomatous formations, but found examples in which the cells lining the acini were columnar in type, and in two instances showed *goblet cells and mucin in the lumen*.

These two cases were apparently on the borderline of malignancy, and no doubt if the patients had lived longer would have become fully formed colloid cancer without the intermediate stage of a macroscopically visible adenoma of the simple type.

The small proportion of cases of colloid cancer with their distribution restricted to the wall of the bladder harmonizes with the rarity of the discovery of these columnar and goblet cells in the routine examination of the urachus. The epithelium in the vast majority of cases attains only to the cubical or low cylindrical stage, and if it goes on to tumour formation, the tumours will in the first instance be simple fibro-adenomata.

EPITHELIAL TUMOURS ARISING ABOVE BETWEEN THE URACHUS AND THE UMBILICUS FROM URACHAL RESTS.

The class of growth which arises above the normal limits of the urachus—that is, in the upper two-thirds of the interval between the umbilicus and the symphysis—have already been referred to (pp. 431, 432). Most of these are mesodermal neoplasms, fibromyomata, fibromata, and sarcomata. They are not tumours of the urachus, but of the fibrous and muscular structures in the space of Retzius. A more interesting, though rarer, variety which occurs in this region must now be considered. These are epithelial tumours, and as in the case of similar types about the bladder apex, five different varieties can be made out: (1) Where adenomatous structures are found on routine examination; (2) Where simple fibro-adenomatous types of larger size have been found clinically; (3) Cases of simple adenocarcinoma; (4) Cases of 'mixed tumours' of unusual structure; (5) Fully developed colloid cancer (adenocarcinoma).

In the whole of the literature only one case each of Groups 3, 4, and 5 has

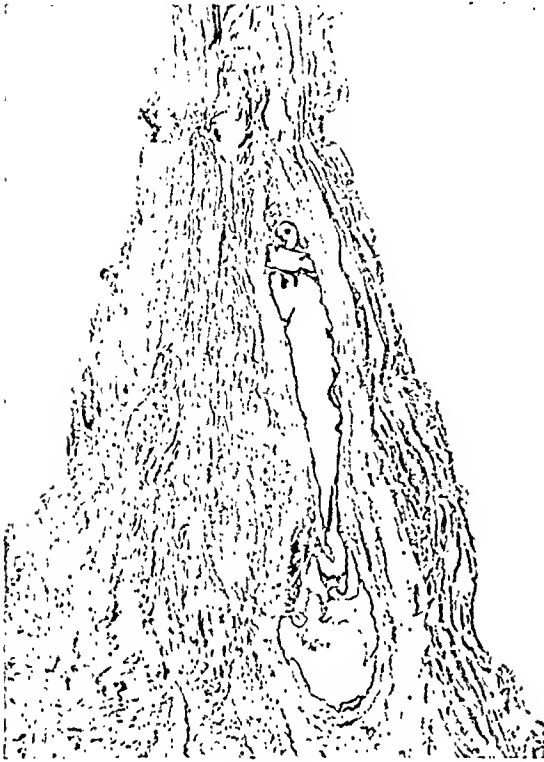


FIG. 349.—Low-power longitudinal section between the umbilicus and the urachus in a child of three (same case as Fig. 328), to show an adenomatous structure, probably derived from cells left during the descent of the urachus.

An adenomatous structure was found, consisting in the main of two narrow cysts with their long axes from above down (Fig. 349). At the upper pole and between the two cysts were glandular-like spaces. A high-power view of the active area in the upper region of the structure is shown in Fig. 350. The cyst wall is covered with medium cylindrical epithelial cells whose nuclei are not regularly basal. The acini are also lined by regular low cylindrical epithelium, but in parts there is more than one layer. There are no goblet cells. The cavity has probably been formed by the amalgamation of two cysts. Solid columns of cells and small cysts may be seen at the poles and at various parts of the circumference. In some respects the structure

been reported, and all without reference to the etiology here suggested. The conclusion that these growths arose from urachal rests is reached on the following grounds: (a) From the nature of their structure, so similar to proved urachal types; (b) From their position, in the cases of those found below the umbilicus and above the urachus; (c) By a process of exclusion, as they do not conform to growths that could have arisen from any other structure in the region.

1. Adenomatous Structures found on Routine Examination of Specimens.—

A female, 3 years of age, died of tuberculous meningitis. At the post-mortem examination no other lesion except the tuberculous ones were found. The urachus attached to the two umbilical arteries formed an anchor-like arrangement some 3 cm. above the bladder, connected to the umbilicus by a well-marked ligamentum commune (see Fig. 328).



FIG. 350.—High-power view of part of same section.

resembles an endometrioma. Its general and detailed appearance almost certainly identifies it with similar adenomata as seen at the bladder end of the urachus, while its narrowness and position along the path of urachal descent suggest that it arose from cells shed by the urachal canal. It begins some distance below the umbilicus, and stops short several centimetres above the apex of the urachus. The latter point was verified by serial sections in a downward direction.

The absence of a *membrana propria* is apparent. This feature distinguishes in general the urachal adenomata from those derived from the omphalo-mesenteric duct.

2. Fibro-adenoma (at or near the umbilicus).—Many cases have been reported of adenomata at the umbilicus which correspond closely in structure to urachal tumours. In some cases the tumour was an adenocarcinoma of the rectal type.

In Ehrlich's case there was present at the umbilicus a tumour 3 cm. in diameter. On microscopical examination glandular spaces in a moderately firm stroma were seen. These spaces were lined in parts by cubical, in other parts by cylindrical, epithelium. The author's illustrations show the typical simple fibro-adenomatous structure without basal membrane.

The subject of umbilical growths is too complex to use as a basis for a pathological theory in regard to urachal growths. An extensive literature exists, and may be studied in the writings of Cullen,²⁸ Kolaczek,⁶⁸ Küstner,^{71, 72} Ledderhose,⁷⁵ Motz,⁸⁶ Mintz,⁸⁴ Ehrlich,³³ Jores,⁶¹ Ahlfeld,² and Pernice.⁸⁴

3. Adenocarcinoma.—

KOSLOWSKI's case⁶⁹ (published 1903).—

Male, age 55. A swelling was palpable below the umbilicus. It was described as being in size and shape like the human patella. From it two cords passed—a thick and indurated one from the upper part of the growth to the umbilicus, and a thinner one to the urachus.

SPECIMEN.—In the capsule was a small amount of smooth muscle fibre. In the midst of the somewhat delicate stroma were glandular spaces and acini lined by columnar epithelium of the regular type. The picture on the whole resembled a urachal tumour, and the smooth muscle may have been derived from the obliterated umbilical arteries.

Kosowski considers the case one of tumour of the omphalo-mesenteric duct. Khaum, Cullen, and others considered it as urachal.

I have never seen a *membrana propria* outside the acini in the proved urachal tumours, and am very doubtful if this can be classified as one. The presence of a basal membrane and the amount of smooth muscle tissue leads me to think that Kosowski was probably right and the tumour was not associated with urachal rests. Unfortunately mere position is no criterion, for, as Schwarz¹¹⁰ has pointed out, the omphalo-mesenteric duct festoons down below the umbilicus and may be caught up in the abdominal wall inferior to that structure.

4. 'Mixed Tumour'.—

ADAM's case¹ (published in 1929).—

Female, age 43. The patient had a slight elevation of the navel from birth. It started to increase in size shortly before she was examined. The lump was incised, and pieces of tumour commenced to come away. In addition to the primary fungating neoplasm, the growth involved and burst through the abdominal wall half-way

between the navel and the symphysis pubis. The masses at the umbilicus appeared white and translucent. The growths were removed by operation and were found to be anterior to the peritoneum.

SPECIMENS.—The larger tumour, the lower of the two, was 7 cm. in diameter, the smaller one at the umbilicus measured 4×3 cm. Both showed on section a stroma infiltrated by carcinomatous cells, and cysts containing mucinous material. Adam thought the lower tumour was primary and the one at the navel secondary. He described them under the name of 'carcinoma planocellulare cysticum'.

It is clear that both tumours arose above the level of the urachus, and from the history it seems probable that the one at the umbilicus was primary. They resemble in structure the cases of Pendl and Barringer, and were probably derived from urachal rests.

5. Colloid Cancer.—

HUE and JACQUIN'S case³⁹ (published in 1863).—

Male, age 45. The patient noticed a smooth tumour in the middle line below the umbilicus. This was incised. Ten months later there was a fungating mass from the navel to the symphysis pubis.

AUTOPSY.—Rectal muscles completely transformed into a gelatinous mass. The parietal peritoneum had undergone a similar change. The tumour involved the bladder apex, and in continuity with it were two colloid masses in the bladder.

This case was probably one of adenocarcinoma originating in urachal rests left near the umbilicus after the descent of the urachus, the growth afterwards spreading by direct descent to the bladder and invading this organ secondarily. It seems evident from the history that the growth started below the umbilicus and spread to the bladder by direct extension. It was probably a colloid cancer, but that it was not a typical tumour is shown by the fact that Laennec considered it a gelatinous cancer. Virchow a gelatinous myxoma, and J. Müller a gelatinous sarcoma (Brady).

From the case-histories given bearing on neoplasms arising from urachal rests, it is evident that we are not treading on such solid ground as when considering the subject of growths of the urachus itself. Clinical material is scanty, but the evidence provided by routine examinations of specimens and by a consideration of the exact structure of the adenomata of the umbilicus gives a good support for the supposition, probable on theoretical grounds, that the urachus during the almost violent nature of its descent and its strong attachment on each side to the umbilical arteries, may spill some of its cells into the transversalis-peritoneal space, and that these are capable of forming tumours of clinical importance.

SUMMARY.

1. On theoretical grounds, colloid adenocarcinomata resembling rectal growths may arise in the muscular wall of the upper part of the bladder.

2. There is definite evidence, both post-mortem and clinical, that such growths do occur as primary tumours, and that the general tendency is for neoplasms in this region to assume the form of colloid cancer.

3. Primitive cells of the urachus are always present in the muscular wall at the apex of the bladder. While these are totipotent, their bias is to form

tumours of the intestinal type. Such neoplasms have, however, certain distinguishing features of their own.

4. Adenomatous and cystic formations of various kinds are commonly found in connection with the lower end of the urachus, and the connection between these and the colloid cancers can be followed through all the stages.

5. All tumours of the bladder apex must be assumed to be of urachal origin from the point of view of treatment unless the contrary is proved.

6. The treatment is by radical operation, removing the umbilicus and all the tissues between it and the bladder, as well as a large portion of the latter.*

REFERENCES.

- ¹ ADAM, L., "Zwei interessante Bauchwandgeschwülste", *Zentralb. f. d. Gesamte Med.*, 1929, xi, 1159.
- ² AHLFELD, *Arch. f. Gynäkol.*, 1876, ix, 325.
- ³ ALBARRAN, J., *Les Tumeurs de la Vessie*, 1892, 101. Paris: Steinheil.
- ⁴ ALEXANDER, S., "Report of a Case of Papillary Adenocarcinoma of the Bladder", *Jour. Cutan. and Gen.-urin. Dis.*, 1891, ix, 86.
- ⁵ ASCHOFF, "Ein Beitrag zur normalen und pathologischen Anatomie der Schleimhaut der Harnwege und ihrer drüsigen Anhänge", *Virchow's Arch.*, 1895, cxxxviii, 119.
- ⁶ ASCHOFF, *Pathologische Anatomie*, 6th ed.
- ⁷ ASTRALDI, "Polypus of the Bladder", *Rev. de Especialidad*, Buenos Aires, 1928, iii, 757.
- ⁸ AUDRY, C., *Le Mercredi médical*, 1893 (cited by Paschkis).
- ⁹ AVELING, *Brit. Gynaecol. Jour.*, 1886-7, ii, 56.
- ¹⁰ BARRINGER, B. S., "Colloid Adenocarcinoma of the Bladder", *Surg. Gynecol. and Obst.*, 1920, xxx, 86.
- ¹¹ BAYER, RUDOLF, "Ein schleimbildendes Cystadenom der Harnblase", *Virchow's Arch.*, 1909, cxvii, 350.
- ¹² BEGG, R. C., "The Urachus: its Anatomy, Histology and Development", *Jour. of Anat.*, 1930, lxiv, 170.
- ¹³ BEGG, R. C., "The Urachus and Urinary Fistula at the Umbilicus", *Surg. Gynecol. and Obst.*, 1927, xlv, 165.
- ¹⁴ BILLROTH, cited by Lenormant.
- ¹⁵ BLAVET DE BRIGA, C., "Un Cas de Cancer de la Vessie avec Kystes", *Jour. d'Urol.*, 1923, xvi, 29.
- ¹⁶ BLUM, V., "Ueber den Gallertkrebs der Harnblase und seine Beziehungen zur Zystenbildung in der Schleimhaut des Harntraktes", *Wien. med. Woch.*, 1914, lxiv, 610.
- ¹⁷ BLUM, V., "Ueber den Gallertkrebs der Harnblase", *Verhandlung. d. deutsch. Gesell. f. Urol.*, 1912, iii, 481.
- ¹⁸ BRADY, L., "Solid Tumours of the Urachus", *Arch. of Surg.*, 1927, xix, 46.
- ¹⁹ BRIDoux, H., *Amer. Jour. Urol.*, 1913, ix, 514.
- ²⁰ BRIDoux, H., "Contribution à l'Etude générale de l'Adénome vésical", *Thèse de Lyon*, 1898.
- ²¹ BRISTOW, A. T., "Adenocarcinoma of the Bladder", *Long Island Med. Jour.*, 1911, 398.
- ²² BURGER, *Zentralb. f. Gynäkol.*, 1905. Describes Störck and Zuckerkandl's first case.
- ²³ CAHEN, F., "Zur Kasuistik der Blasentumoren", *Virchow's Arch.*, 1888, cxlii, 468.
- ²⁴ CARRARO, Transactions of the First Congress of the Italian Urological Association, Florence, October, 1922, *Zeits. f. urol. Chir.*, 1922-3, ii, 224.
- ²⁵ CASSANELLO, R., "Contribution à l'Etude anatomo-pathologique et clinique de l'Adénome vésical", *Ann. des Mal. des Org. gén.-urin.*, 1908, i, 641.
- ²⁶ CHUTE, A. L., and CROSBIE, A. H., "Mucus Cancer of the Bladder", *Boston Med. and Surg. Jour.*, 1912, clxvii, 583.

*Recently another case of a tumour at the bladder apex and involving the urachus has been described by Lane and Monson (*Brit. Jour. Urol.* 1930, ii, 272).

- ²⁷ CLADO, "Traité des Tumeurs de la Vessie", cited by Lenormant (same case as Marcacci's).
- ²⁸ CULLEN, T. S., *The Umbilicus and its Diseases*, 1916, 637. Philadelphia: W. B. Saunders Co.
- ²⁹ DE KORTE, W. E., "An Adenoma of the Bladder", *Jour. Pathol. and Bacteriol.*, 1918-19, xxii, 319.
- ³⁰ DOIRAN, *Lancet*, 1909, i, 1314.
- ³¹ EGGER, O., "Ueber Blasengeschwülste", *Zeits. f. urol. Chir.*, 1921, vi, 174.
- ³² EHRLICH, D. E., "Gallertkrebs der ekstrophierten Harnblase", *Beitr. z. klin. Chir.*, 1901, xxx, 581.
- ³³ EHRLICH, "Primäres doppelseitiges Mammakarcinom und wahres Nabeladenom", *Arch. f. klin. Chir.*, 1909, lxxxix, 742.
- ³⁴ EISELBERG, "Fall von Karzinom der Schleimhaut einer Blasenektomie", *Wien. klin. Woch.*, 1906, 548. Describes Störck and Zuckerkaudl's third case.
- ³⁵ EWENS, cited by Greig Smith.
- ³⁶ FISCHER, "Die Eiterungen in subumbilicalem Raume", *Sammlung klin. Vorträge*, 1893, Leipsic. Cited by Brady.
- ³⁷ FONTANA, A., "I Tumori epiteliali della Vescica", *Tumori*, 1920, v, No. 15, 185.
- ³⁸ FRANK, T., "Zur Kasuistik der Urachstumoren", *Inaug. Dissert.*, Würzburg, 1893.
- ³⁹ FRISCH und ZUCKERKAUDL, *Handbuch der Urologie*, 1905, ii.
- ⁴⁰ GERAHTY, J. T., "The Results of the Treatment of Bladder Tumors", *Jour. Amer. Med. Assoc.*, 1917, lxix, 1336.
- ⁴¹ GERAHTY, J. T., *Cabot's Modern Urology*, 1918, ii, 187.
- ⁴² GIOUDANO, "Perithelioma of the Bladder", *Minerva Med.*, 1920, ix, pt. 1, 676.
- ⁴³ GÖBEL, C., "Ueber die bei Bilharziakrankheit vorkommende Blasentumoren mit besonderer Berücksichtigung des Karcinoms", *Zeits. f. Krebsforsch.*, 1905, iii, 450.
- ⁴⁴ GOODALL, R. H., "Cystadenoma of Aberrant Prostatic Glands", *Arch. of Pathol.*, 1928, vi, 210.
- ⁴⁵ GREEN, N. W., "Gelatinous Carcinoma of the Bladder", *Ann. of Surg.*, 1915, lxii, 501.
- ⁴⁶ GRIEG, D. M., "Report of a Case of Sarcoma of the Urachus." *Edin. Med. Jour.*, 1927, xxxiv, 425.
- ⁴⁷ HAARE, OTTO, "Ueber den primären Krebs der Harnblase", *Inaug. Dissert.*, Freiburg, 1895.
- ⁴⁸ HAGER, A., "Ekstrophia Vesicae urinariae mit Adenocarcinoma gelatiniforme", *Munch. med. Woch.*, 1910, 2301.
- ⁴⁹ HAGNER, "Report of a Case of Fibro-adenoma in the Bladder Wall", *Amer. Jour. Urol.*, 1907, iii, 414.
- ⁵⁰ HARTMANN, "Quelques Réflexions à propos de 47 Opérations pour Tumeurs de la Vessie", *Travaux de Chir. anatom-clin.*, 4th s., 1922.
- ⁵¹ HARTMANN, personal communication to Lenormant.
- ⁵² HECTOR, C. M., personal communication.
- ⁵³ HEINEKE, *Munch. med. Woch.*, 1913, cited by Pinesohn.
- ⁵⁴ HERBST, R. H., and THOMPSON, A., "Adenocarcinoma of the Bladder", *Amer. Jour. Surg.*, 1922, xxxvi, 4.
- ⁵⁵ HERZEL, cited by Krompever.
- ⁵⁶ HERMANN, L., "Neoplasm of the Trigonum Vesicae. A Probable Instance of Hypertrophic Changes in Aberrant Prostatic Tissue", *Jour. of Urol.*, 1928, xix, 291.
- ⁵⁷ HOFFMANN, C. E. E., "Zur pathologisch-anatomischen Veränderung des Harnstrangs", *Arch. f. Heilk.*, 1870, xi, 373.
- ⁵⁸ HORN, W., and ORATOR, V., "Zur Frage der Prostatahypertrophie", *Frankf. Zeits. f. Pathol.*, 1922, xxviii, 340.
- ⁵⁹ HUE and JACQUIN, "Cancer colloïde de l'Ombilic et de la Paroi abdominale antérieure ayant envahi la Vessie", *Union méd.*, 1863, vi, 418.
- ⁶⁰ HÜTTENBRENNER, "Ueber den histologischen Bau des Sarkomphalus bei Kinder", *Zeits. f. Heilk.*, 1882, 1.
- ⁶¹ JONES, "Ein Nabeladenom", 1899, cited by Mintz.
- ⁶² JUDS, S., "Adenomyoma presenting a Tumor of the Bladder", *Surg. Clin. N. America*, 1921, i, 1271.
- ⁶³ KALTENBACH, "Extirpation eines papillären Adenoms der Harnblase von der Scheide aus", *Arch. f. klin. Chir.*, 1884, xxx, 659.
- ⁶⁴ KANAMORI, "Zwei Fälle von Blasentumoren", *Virchow's Arch.*, 1897, cxlvii, 119.
- ⁶⁵ KHAUM, E., "Ueber ein primäres Karcinom des Urachus", *Wien. klin. Woch.*, 1916, cxxx, 131.
- ⁶⁶ KIELLEUTNER, L., "Blasenscheiteltumor-Urachuskarcinom", *Zeits. f. Urol.*, 1929, xxxiii, 519.

- ⁶⁷ KLOPP, E. J., "Patent Urachus with Sarcoma developing in the Wall", *Ann. of Surg.*, 1921, lxxiii, 643.
- ⁶⁸ KOLACZEK, *Virchow's Arch.*, lxxix, 537.
- ⁶⁹ KOSŁOWSKI, B. S., "Ein Fall von wahrem Nabeladenom", *Deut. Zeits. f. Chir.*, 1903, lxxix, 469.
- ⁷⁰ KROMPEYER, *Beitr. z. pathol. Anat. u. z. allgem. Pathol.*, 1908, xlv, 88. Describes Herezel's case.
- ⁷¹ KÜSTNER, O., "Notiz über den Bau des Fungus umbilici", *Arch. f. Gynäkol.*, 1876, ix, pt. 3.
- ⁷² KÜSTNER, O., "Das Adenom und die Granulationsgeschwülste am Nabel des Kindes", *Virchow's Arch.*, 1877, lxxix, pt. 2, 286.
- ⁷³ LAVAUX, *Chirurgie contemporaine des Org. gén.-urin.*, 1892. Cited by Paschakis.
- ⁷⁴ LAVENANT, A., "Epithélioma colloïde d'Origine allantoïdienne du Sommet de la Vessie", *Jour. d'Urol.*, 1924, xvii, 43.
- ⁷⁵ LEDDERHOSE, "Die chirurgischen Erkrankungen der Bauchdecken", *Deut. Chir.*, 1890.
- ⁷⁶ LENDORF, "Beiträge zur Histologie der Harnblasenschleimhaut", *Anatomic.* Cited by Zueckerkandl.
- ⁷⁷ LENORMANT, C., "Sur un Cas de Tumeur mixte (Epithéliosarcome) de la Vessie", *Jour. d'Urol.*, 1922, xiv, 273.
- ⁷⁸ LONG, J. H., "Dermoid Cyst of the Urachus", *Surg. Clin. N. America*, 1927, vii, 943.
- ⁷⁹ LUBARSCH, O., "Ueber Cysten der ableitenden Harnwege", *Arch. f. microscop. Anat.*, 1893, xli, 303.
- ⁸⁰ LUSCHKA, "Ueber den Bau des menschlichen Harnstranges", *Virchow's Arch.*, 1862, xxiii, 1.
- ⁸¹ MANDELBAUM, cited by Ewing in the case of Barringer.
- ⁸² MARCACC, cited by Lenormant, loc. cit.
- ⁸³ MICHIN, W. A., "Zur Kasuistik der aus den Resten des Ductus omphalo-mesentericus entwickelnden malignen Neubildungen", *Virchow's Arch.*, 1912, ccix, 47.
- ⁸⁴ MINTZ, W., "Das Nabeladenom", *Arch. f. klin. Chir.*, 1909, lxxxix, 385.
- ⁸⁵ MINTZ, W., "Das wahre Adenom des Nabels", *Deut. Zeits. f. Chir.*, 1899, li, 545.
- ⁸⁶ MOLTZ, "Quatre Cas d'Adénome de la Vessie", *Ann. des Mal. des Org. gén.-urin.*, 1898, xvi, 1228.
- ⁸⁷ NICHOLSON, G. W., "Heteromorphoses in the Human Body", *Guy's Hosp. Rep.*, 1922, 97.
- ⁸⁸ NOTTHART, A., "Ueber die Entstehung des Karcinoms", *Deut. Arch. f. klin. Med.*, 1895, liv, 555.
- ⁸⁹ NUDER, J. F., "Ein Fall von Carcinoma urachi", *Virchow's Arch.*, 1925, ccliv, 70.
- ⁹⁰ PASCHAKIS, R., "Ueber Adenome der Harnblase", *Zeits. f. urol. Chir.*, 1926-7, xxi, 315.
- ⁹¹ PASCHAKIS, R., *Wien. klin. Woch.*, 1910, No. 47, 1695.
- ⁹² PAVONI, "Deux Cas de Tumeurs de la Vessie." Given at the 11th session of l'Association française d'Urologie. Cited by Cassanello.
- ⁹³ PENDEL, F., "Gallertkrebs einer Urachuseyste", *Beitr. z. klin. Chir.*, 1914.
- ⁹⁴ PERNICI, *Die Nabelgeschwülste*, Halle. Cited by Ehrlich.
- ⁹⁵ PINESON, A., "Entstehung eines Gallertkrebses auf Grund ortswidrigen Epithels in der Harnblase", *Virchow's Arch.*, 1921, cxxxii, 350.
- ⁹⁶ PINILERO, G., *Clin. Urolog.*, Barcelona. Cited by Keilleuthner.
- ⁹⁷ POSNER, "Ein Fall von primärem Carcinom der Harnblase", *Berl. klin. Woch.*, 1883, 392.
- ⁹⁸ PEREMESCHKO, cited by Michin.
- ⁹⁹ RANDALL, A., "Disease of the Urachus. Report of Two Cases", *Trans. Amer. Assoc. Gen.-urin. Surgeons*, 1926, ix, 211.
- ¹⁰⁰ RANKIN, F. W., and PANKEN, B., "Tumors of the Urachus", *Surg. Gynecol. and Obst.*, 1926, xlii, 19.
- ¹⁰¹ RAUENBUSCH, L., "Ueber Gallertkrebs der Harnblase", *Virchow's Arch.*, 1905, clxxxii, 132.
- ¹⁰² ROCHET and MARTEL, *Gaz. hebdom. de Méd. et Chir.* Cited by Cassanello.
- ¹⁰³ ROTTER, *Zentralb. f. Chir.*, 1897, xxvi, 604.
- ¹⁰⁴ ROTTER, *Deut. med. Woch.*, 1898, No. 7, 10.
- ¹⁰⁵ SACCHI, "Cistadenoma della Vesica", *Arch. Ital. di Chir.*, 1923, vii, 161 (Extract in *Zeits. f. urol. Chir.*, 1923-4, xiv, 227).
- ¹⁰⁶ SAUTER, R., "Ein Fall von Gallertkarzinom der Harnblase", *Inaug. Dissert.*, München, 1898.
- ¹⁰⁷ SCHMIDTMANN, M., "Zur Kenntniss seltener Krebsformen", *Virchow's Arch.*, 1919, cxxxvi, 100.
- ¹⁰⁸ SCHOLL, J., "Histology and Mortality in Cases of Tumor of the Bladder", *Surg. Gynecol. and Obst.*, 1922, xxxiv, 189.

- ¹⁰⁹ SCHRIDDE, *Die Entwicklungsgeschichte des menschlichen Speiserohrenepithels*, 1907, Wiesbaden.
- ¹¹⁰ SCHWARZ, E., "Das Carcinom des Urachus", *Beitr. z. klin. Chir.*, 1912, lxxviii, 287.
- ¹¹¹ SHARP, H. C., "Primary Colloid Cancer of the Bladder", *Trans. Pathol. Soc. Lond.*, 1896, March 5.
- ¹¹² SIMON, cited by Kaltenbach.
- ¹¹³ SMITH, GREG, *Abdominal Surgery*, 1897, ii, 1070 (2 cases).
- ¹¹⁴ SPEHJING, cited by Chute and Crosbie.
- ¹¹⁵ STÖRCK, "Beiträge zur Pathologie der Schleimhaut der harleitenden Wege", *Beitr. z. pathol. Anat.*, 1899, xxvi, 367.
- ¹¹⁶ STÖRCK und ZUCKERKANDL, "Ueber Cystitis glandularis und den Drüsenkrebs der Harnblase", *Zeits. f. Urol.*, 1907, i, 2 and 133.
- ¹¹⁷ STÖRCK und ZUCKERKANDL, *Wien. klin. Woch.*, 1906, xx, 548.
- ¹¹⁸ SUCHANNEK, H., "Beiträge zur Kenntniss des Urachus", *Dissert.*, Königsberg, 1879.
- ¹¹⁹ THOMAS, B. A., *Jour. of Urol.*, 1928, xix, 291 (discussion on Hermann's paper).
- ¹²⁰ UTRAU, *Jour. d'Urol.*, 1924, xviii, 472.
- ¹²¹ VERHOOGEN, J., "Néoplasmes de la Vessie", *Encyclopédie franç. d'Urol.*, 1913, iv, 470.
- ¹²² VILLA, F., "Tumor dell' Uraco", *Gaz. med. di Milano*, 1902, i, 65.
- ¹²³ VINCHOW, *Virchow's Arch.*, v, 216.
- ¹²⁴ VON BRUNN, A., "Ueber drüsenähnliche Bildungen in der Schleimhaut des Nierenbeckens, des Ureters und der Harnrohre beim Menschen", *Arch. f. mikroskop. Anat.*, 1893, xli, 294.
- ¹²⁵ VONCKEN und CAMMERMEYER, *Arch. med. Belges*, 1927, 80 (2 cases); *Jour. de Chir. et Ann. Soc. Belge*, 1927, xxi, 315.
- ¹²⁶ VON LIMMECK, R., "Zur Kenntniss der Epitheleysten der Harnblase und der Ureteren", *Zeits. f. Heilk.*, 1887, viii, 55.
- ¹²⁷ WÄGELER, F., "Zur Histogenese der Nabeladenome nebst einen kasuistischen Beitrag", *Zeits. f. Pathol.*, 1913, xiv, 367.
- ¹²⁸ WÄGELER, F., "Nabeladenome", *Deut. med. Woch.*, 1913, No. 8, 2.
- ¹²⁹ WATSON, F., "The Operative Treatment of Tumours of the Bladder", *Ann. of Surg.*, 1905, xlii, 805.
- ¹³⁰ WESSON, "Anatomical, Embryological, and Physiological Studies of the Trigone and Neck of the Bladder", *Jour. of Urol.*, 1920, iv, 279.
- ¹³¹ WITTSACH, "Ein primäres Adenom der Harnblase beim Manne", *Zentralb. f. die Krankh. der Haru u. Sex.-organ.*, 1894, v, 458.
- ¹³² WITZ, J. B., "Ueber Urachus und Urachuscysten", *Virchow's Arch.*, 1883, xcii, 387.
- ¹³³ ZÄRSCH, C., "Zur Statistik des Carcinoma Vesicae", *Inaug. Dissert.*, München, 1887.
- ¹³⁴ ZUCKERKANDL, O., "Umwandlung des Blasenepithels in sezernierendes Zylinder-epithels", *Zeits. f. Urol.*, 1911, v, 622.

**STUDIES ON HIGH INTESTINAL OBSTRUCTION:
THE ADMINISTRATION OF SALINE AND OTHER SUBSTANCES BY
ENTEROSTOMY BELOW THE SITE OF OBSTRUCTION.**

BY J. C. ARMOUR, T. G. BROWN, D. M. DUNLOP, T. C. MITCHELL,
H. H. SEARLS, AND C. P. STEWART,

FROM THE DEPARTMENTS OF EXPERIMENTAL SURGERY AND BACTERIOLOGY, UNIVERSITY OF EDINBURGH,
AND THE CLINICAL LABORATORIES, ROYAL INFIRMARY, EDINBURGH.

INTRODUCTION.

This investigation was undertaken in an attempt to determine the cause of death in high intestinal obstruction, and to elaborate if possible a means of preventing death after the establishment of the condition. At the outset it is to be emphasized that the study was confined to simple high obstruction, the questions—entirely distinct, as we believe—of strangulation and ‘closed loops’ not being considered.

Opinion as to the cause of death in high intestinal obstruction is more or less sharply divided. The oldest theory is that death is due to the absorption of some toxic substance formed above the site of the obstruction. The origin of this hypothetical toxin has been the subject of much discussion. There are many who attribute it to bacterial putrefaction of the intestinal contents above the obstruction, where, indeed, both aerobic and anaerobic organisms may be present in large numbers. In particular, attention has been drawn to the possible importance of *B. welchii* in the production of symptoms in intestinal obstruction by the work of Williams.¹ Struck by the clinical resemblance of some of the symptoms of obstruction to those of gas gangrene, he began to investigate, both qualitatively and quantitatively, the occurrence of *B. welchii* in the contents of the obstructed intestine, and to administer antitoxin as a therapeutic measure. As a result of combined experimental work and clinical study he finally suggested that the absorption of *B. welchii* toxin was, in part at least, responsible for the symptoms of obstruction. He also brought forward evidence to show that the proliferation of *B. welchii* and the presence of its toxin could apparently be demonstrated in the human subject and in dogs. The therapeutic use of anti-gas-gangrene serum showed, in Williams’s cases, a fall in the mortality-rate from 24·8 per cent to 9·3 per cent. His work excited considerable interest, and serum has been extensively used in the treatment of cases of acute intestinal obstruction.

According to Whipple,² however, a toxic proteose is formed in the intestinal mucous membrane itself, part being excreted into the lumen and part passing into the circulation. It is difficult, however, to explain along these lines or along any lines involving the production of a toxin the fact that death follows an obstruction just below the pylorus with great rapidity,

whereas the time of survival is increased when the obstruction is lower down. It would seem, if the toxic theory is correct, that death should be more rapid when there is a greater length of bowel available for the absorption of the toxin than when the absorbing surface is small. Besides those already mentioned, various other sources of the hypothetical toxin have been suggested, including the pancreatic and biliary secretions.

A more recent theory is that death is due to a profound alteration in the chemical composition of the blood—loss of chloride, dehydration, and alkalosis—which reflects upon the physical and chemical processes of the whole body. The chief supporters of this view are Gamble and Ross.³ Haden and Orr⁴ subscribed to the toxin theory, but held that chloride was important in rendering the toxin harmless by a process akin to neutralization. In the course of their experiments they succeeded in keeping obstructed dogs alive for from twenty-one to twenty-eight days by daily subcutaneous injection of 500 c.c. of 0.9 per cent saline. During the progress of the present work White and Fender⁵ reported that they had maintained a dog alive and well for twenty-eight days by re-injecting the gastric contents into the bowel below the obstruction. Matsukura⁶ succeeded in prolonging the life of a dog with high obstruction to thirty-three days by injection of sodium chloride and water into an enterostomy below the obstruction. Death at the end of these periods might well be attributed to starvation. Among other causes of death in high intestinal obstruction which have been suggested are that it is due to disordered function of duodenal secretions⁷ and hepatic insufficiency.⁸

In the experiments to be described in this paper an attempt was made to determine whether, by administration of saline together with energy-producing foodstuffs, life could be sufficiently prolonged to eliminate the possibility of toxæmia as a cause of death. The method of administration—below the site of obstruction—would appear to prevent the possibility of the saline's acting merely as a diluent or neutralizing agent of a toxin produced in the obstructed part of the alimentary tract. Further, on the assumption that the toxin, if toxin there be, is of bacterial origin, experiments were made to find if any correlation existed between the bacterial growth above the obstruction and the condition of the animal.

EXPERIMENTAL METHODS.

Operative Technique.—Healthy dogs of one to three years of age were chosen. On the morning of operation food, but not water, was withheld. Anaesthesia was secured by the open-ether method. The abdomen was opened by a right paramesial incision extending from the costal margin to the umbilical level, and the intestine was brought out into the wound. An obstruction by the 'severed-loop' method was effected at the tail of the pancreas below the lowest pancreatic duct and 10 to 15 cm. from the pylorus. This was the usual site of the obstruction. In a number of cases different positions were chosen, and these are mentioned in the reports on the particular dogs concerned. A transverse incision through the serous coat was made round the duodenum; the muscular coat was divided and reflected by blunt dissection into a proximal and a distal cuff. Two silk purse-string sutures, $\frac{1}{2}$ cm. apart, were then inserted in the submucous coat, and the gut was divided between them. The proximal end was invaginated by further purse-string sutures in the seromuscular coat, and was restored to the abdomen. A Pezzer catheter (size 6),

inserted by Senn's method into the distal opening, was brought out through a stab incision about 3 cm. to the right of the mid-line at the level of the umbilicus. The free end of the distal loop of gut was anchored by sutures to the peritoneum of the anterior abdominal wall, omentum being used to reinforce the apposition. The abdominal wound was closed in layers. The integrity of the obstruction and the absence of infection were, in each case, established post mortem, except in the case of *Dog 6*, which is still living, and in which the condition was inspected at the time of re-establishing the continuity of the gastro-intestinal tract.

Pre-operative Treatment.—The animals were kept in special metabolism cages, and observations were carried out for three or four days prior to the operation. Water was freely allowed, as was food consisting of a mash made of boiled meat, dog-biscuit, and rice. Daily measurement of the body weight was made after the aspiration of the fasting gastric contents in the morning. The fasting contents, withdrawn into sterile containers by means of a vacuum pump, were examined bacteriologically and chemically, as was a second sample of gastric contents similarly withdrawn three-quarters of an hour after a gruel meal (10 c.c. per kilo. body weight). The twenty-four hour urine and stool were collected, and an oxalated specimen of blood was taken.

Post-operative Treatment.—The animal was again kept in the metabolism cage, and was weighed daily. Vomiting, with consequent contamination of urine or stool, was prevented as far as possible by aspiration of the gastric contents two or three times daily, the total amount collected in the twenty-four hours being recorded. Chemical examination of the mixed sample was made daily, and specimens were collected twice a week. The urine passed in twenty-four hours was measured and submitted to chemical analysis. Specimens of stool during the whole post-operative period were mixed and the chloride content was determined. Post mortem the chloride concentration in the skeletal muscles was estimated.

No food or water was administered by mouth as a rule, but water was sometimes given to cleanse the mouth and throat, or to wash out the stomach, being withdrawn immediately afterwards. Enterostomy feeds were given thrice daily, the first just after aspiration in the morning, the second in the afternoon, and the third at night. In feeding, the apparatus shown in *Fig. 351* was used. The temperature and rate of flow of the fluid were under control. A manometer connected with the enterostomy tube indicated the pressure in the gut at the site of entry of the fluid into the lumen, thus giving a valuable guide as to the rate at which the fluid should be allowed to enter. Normally, with a manometer tube of bore 0.6 cm., the column varied from 7 to 12 cm. in height above the level of the enterostomy, rising with inspiration and falling with expiration. The optimal rate of flow was approximately a litre in one and a half hours. A few seconds' warning of impending vomiting was given by a steady and rapid rise of water in the manometer, and by immediately stopping the flow of the fluid vomiting could be avoided. Atonicity of the intestine was shown by an exaggerated expiratory excursion of the manometer column: under these conditions it was desirable to reduce the rate of flow. If, however, the mean pressure fell permanently, the rate of flow could conveniently be increased. Mucus in the enterostomy tube had a ball-valve-like action; the column began to rise with inspiration in the ordinary way, but was abruptly arrested, falling suddenly again towards the end of expiration. By slightly increasing the fluid pressure for a few moments the tube could be cleared.

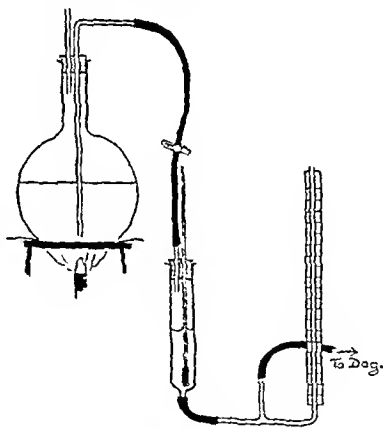


FIG. 351.—Apparatus for continuous feeding by enterostomy.

- ¹⁰⁹ SCHIRIDDE, *Die Entwicklungsgeschichte des menschlichen Speiseröhrenepithels*, 1907, Wiesbaden.
- ¹¹⁰ SCHWARZ, E., "Das Carcinom des Uraehus", *Beitr. z. klin. Chir.*, 1912, lxxviii, 287.
- ¹¹¹ SHARP, H. C., "Primary Colloid Cancer of the Bladder", *Trans. Pathol. Soc. Lond.*, 1896, March 5.
- ¹¹² SIMON, cited by Kaltenbaeh.
- ¹¹³ SMITH, GMEIG, *Abdominal Surgery*, 1897, ii, 1070 (2 cases).
- ¹¹⁴ SPENLING, cited by Chute and Crosbie.
- ¹¹⁵ STÖRCK, "Beiträge zur Pathologie der Schleimhaut der harleitenden Wege", *Beitr. z. pathol. Anat.*, 1899, xxvi, 367.
- ¹¹⁶ STÖRCK and ZUCKERKANDL, "Ueber Cystitis glandularis und den Drüsenkrebs der Harnblase", *Zeits. f. Urol.*, 1907, i, 2 and 133.
- ¹¹⁷ STÖRCK and ZUCKERKANDL, *Wien. klin. Woch.*, 1906, xx, 548.
- ¹¹⁸ SUCHANNEK, H., "Beiträge zur Kenntniss des Urachus", *Dissert.*, Königsberg, 1879.
- ¹¹⁹ THOMAS, B. A., *Jour. of Urol.*, 1928, xix, 291 (discussion on Hermann's paper).
- ¹²⁰ UTEAU, *Jour. d'Urol.*, 1924, xviii, 472.
- ¹²¹ VERHOOGEN, J., "Néoplasmes de la Vessie", *Encyclopédie franç. d'Urol.*, 1913, iv, 470.
- ¹²² VILLA, F., "Tumor dell' Uraco", *Gaz. med. di Milano*, 1902, i, 65.
- ¹²³ VIRCHOW, *Virchow's Arch.*, v, 216.
- ¹²⁴ VON BRUNN, A., "Ueber drüsenähnliche Bildungen in der Schleimhaut des Nierenbeckens, des Ureters und der Harnrohre beim Menschen", *Arch. f. mikroskop. Anat.*, 1893, xli, 294.
- ¹²⁵ VONCKEN and CAMBRESIEN, *Arch. med. Belges*, 1927, 80 (2 cases); *Jour. de Chir. et Ann. Soc. Belg.*, 1927, xxi, 315.
- ¹²⁶ VON LIMBECK, R., "Zur Kenntniss der Epithelcysten der Harnblase und der Ureteren", *Zeits. f. Heilk.*, 1887, viii, 55.
- ¹²⁷ WÄGGLER, F., "Zur Histogenese der Nabeladenome nebst einen kasuistischen Beitrag", *Zeits. f. Pathol.*, 1913, xiv, 367.
- ¹²⁸ WÄGGLER, F., "Nabeladenome", *Deut. med. Woch.*, 1913, No. 8, 2.
- ¹²⁹ WATSON, F., "The Operative Treatment of Tumours of the Bladder", *Ann. of Surg.*, 1905, xlii, 805.
- ¹³⁰ WESSON, "Anatomical, Embryological, and Physiological Studies of the Trigone and Neck of the Bladder", *Jour. of Urol.*, 1920, iv, 279.
- ¹³¹ WITTSACH, "Ein primäres Adenom der Harnblase beim Manne", *Zentralb. f. die Krankh. der Harn u. Sex.-organ.*, 1894, v, 458.
- ¹³² WITZ, J. B., "Ueber Urachus und Urachuscysten", *Virchow's Arch.*, 1883, xcii, 387.
- ¹³³ ZAUSCH, C., "Zur Statistik des Carcinoma Vesicæ", *Inaug. Dissert.*, München, 1887.
- ¹³⁴ ZUCKERKANDL, O., "Umwandlung des Blasenepithels in sezernierendes Zylinder-epithels", *Zeits. f. Urol.*, 1911, v, 622.

STUDIES ON HIGH INTESTINAL OBSTRUCTION:**THE ADMINISTRATION OF SALINE AND OTHER SUBSTANCES BY
ENTEROSTOMY BELOW THE SITE OF OBSTRUCTION.**

By J. C. ARMOUR, T. G. BROWN, D. M. DUNLOP, T. C. MITCHELL,
H. H. SEARLS, AND C. P. STEWART,

FROM THE DEPARTMENTS OF EXPERIMENTAL SURGERY AND BACTERIOLOGY, UNIVERSITY OF EDINBURGH,
AND THE CLINICAL LABORATORIES, ROYAL INFIRMARY, EDINBURGH.

INTRODUCTION.

THIS investigation was undertaken in an attempt to determine the cause of death in high intestinal obstruction, and to elaborate if possible a means of preventing death after the establishment of the condition. At the outset it is to be emphasized that the study was confined to simple high obstruction, the questions—entirely distinct, as we believe—of strangulation and ‘closed loops’ not being considered.

Opinion as to the cause of death in high intestinal obstruction is more or less sharply divided. The oldest theory is that death is due to the absorption of some toxic substance formed above the site of the obstruction. The origin of this hypothetical toxin has been the subject of much discussion. There are many who attribute it to bacterial putrefaction of the intestinal contents above the obstruction, where, indeed, both aerobic and anaerobic organisms may be present in large numbers. In particular, attention has been drawn to the possible importance of *B. welchii* in the production of symptoms in intestinal obstruction by the work of Williams.¹ Struck by the clinical resemblance of some of the symptoms of obstruction to those of gas gangrene, he began to investigate, both qualitatively and quantitatively, the occurrence of *B. welchii* in the contents of the obstructed intestine, and to administer antitoxin as a therapeutic measure. As a result of combined experimental work and clinical study he finally suggested that the absorption of *B. welchii* toxin was, in part at least, responsible for the symptoms of obstruction. He also brought forward evidence to show that the proliferation of *B. welchii* and the presence of its toxin could apparently be demonstrated in the human subject and in dogs. The therapeutic use of anti-gas-gangrene serum showed, in Williams’s cases, a fall in the mortality-rate from 24·8 per cent to 9·3 per cent. His work excited considerable interest, and serum has been extensively used in the treatment of cases of acute intestinal obstruction.

According to Whipple,² however, a toxic proteose is formed in the intestinal mucous membrane itself, part being excreted into the lumen and part passing into the circulation. It is difficult, however, to explain along these lines or along any lines involving the production of a toxin the fact that death follows an obstruction just below the pylorus with great rapidity,

whereas the time of survival is increased when the obstruction is lower down. It would seem, if the toxic theory is correct, that death should be more rapid when there is a greater length of bowel available for the absorption of the toxin than when the absorbing surface is small. Besides those already mentioned, various other sources of the hypothetical toxin have been suggested, including the pancreatic and biliary secretions.

A more recent theory is that death is due to a profound alteration in the chemical composition of the blood—loss of chloride, dehydration, and alkalosis—which reflects upon the physical and chemical processes of the whole body. The chief supporters of this view are Gamble and Ross.³ Haden and Orr⁴ subscribed to the toxin theory, but held that chloride was important in rendering the toxin harmless by a process akin to neutralization. In the course of their experiments they succeeded in keeping obstructed dogs alive for from twenty-one to twenty-eight days by daily subcutaneous injection of 500 c.c. of 0.9 per cent saline. During the progress of the present work White and Fender⁵ reported that they had maintained a dog alive and well for twenty-eight days by re-injecting the gastric contents into the bowel below the obstruction. Matsukura⁶ succeeded in prolonging the life of a dog with high obstruction to thirty-three days by injection of sodium chloride and water into an enterostomy below the obstruction. Death at the end of these periods might well be attributed to starvation. Among other causes of death in high intestinal obstruction which have been suggested are that it is due to disordered function of duodenal secretions⁷ and hepatic insufficiency.⁸

In the experiments to be described in this paper an attempt was made to determine whether, by administration of saline together with energy-producing foodstuffs, life could be sufficiently prolonged to eliminate the possibility of toxæmia as a cause of death. The method of administration—below the site of obstruction—would appear to prevent the possibility of the saline's acting merely as a diluent or neutralizing agent of a toxin produced in the obstructed part of the alimentary tract. Further, on the assumption that the toxin, if toxin there be, is of bacterial origin, experiments were made to find if any correlation existed between the bacterial growth above the obstruction and the condition of the animal.

EXPERIMENTAL METHODS.

Operative Technique.—Healthy dogs of one to three years of age were chosen. On the morning of operation food, but not water, was withheld. Anæsthesia was secured by the open-ether method. The abdomen was opened by a right paramesial incision extending from the costal margin to the umbilical level, and the intestine was brought out into the wound. An obstruction by the 'severed-loop' method was effected at the tail of the pancreas below the lowest pancreatic duct and 10 to 15 cm. from the pylorus. This was the usual site of the obstruction. In a number of cases different positions were chosen, and these are mentioned in the reports on the particular dogs concerned. A transverse incision through the serous coat was made round the duodenum; the muscular coat was divided and reflected by blunt dissection into a proximal and a distal cuff. Two silk purse-string sutures, $\frac{1}{2}$ cm. apart, were then inserted in the submucous coat, and the gut was divided between them. The proximal end was invaginated by further purse-string sutures in the seromuscular coat, and was restored to the abdomen. A Pezzer catheter (size 6),

inserted by Senn's method into the distal opening, was brought out through a stab incision about 3 cm. to the right of the mid-line at the level of the umbilicus. The free end of the distal loop of gut was anchored by sutures to the peritoneum of the anterior abdominal wall, omentum being used to reinforce the apposition. The abdominal wound was closed in layers. The integrity of the obstruction and the absence of infection were, in each case, established post mortem, except in the case of *Dog 6*, which is still living, and in which the condition was inspected at the time of re-establishing the continuity of the gastro-intestinal tract.

Pre-operative Treatment.—The animals were kept in special metabolism cages, and observations were carried out for three or four days prior to the operation. Water was freely allowed, as was food consisting of a mash made of boiled meat, dog-biscuit, and rice. Daily measurement of the body weight was made after the aspiration of the fasting gastric contents in the morning. The fasting contents, withdrawn into sterile containers by means of a vacuum pump, were examined bacteriologically and chemically, as was a second sample of gastric contents similarly withdrawn three-quarters of an hour after a gruel meal (10 c.c. per kilo. body weight). The twenty-four hour urine and stool were collected, and an oxalated specimen of blood was taken.

Post-operative Treatment.—The animal was again kept in the metabolism cage, and was weighed daily. Vomiting, with consequent contamination of urine or stool, was prevented as far as possible by aspiration of the gastric contents two or three times daily, the total amount collected in the twenty-four hours being recorded. Chemical examination of the mixed sample was made daily, and specimens were collected twice a week. The urine passed in twenty-four hours was measured and submitted to chemical analysis. Specimens of stool during the whole post-operative period were mixed and the chloride content was determined. Post mortem the chloride concentration in the skeletal muscles was estimated.

No food or water was administered by mouth as a rule, but water was sometimes given to cleanse the mouth and throat, or to wash out the stomach, being withdrawn immediately afterwards. Enterostomy feeds were given thrice daily, the first just after aspiration in the morning, the second in the afternoon, and the third at night. In feeding, the apparatus shown in Fig. 351 was used. The temperature and rate of flow of the fluid were under control. A manometer connected with the enterostomy tube indicated the pressure in the gut at the site of entry of the fluid into the lumen, thus giving a valuable guide as to the rate at which the fluid should be allowed to enter. Normally, with a manometer tube of bore 0.6 cm., the column varied from 7 to 12 cm. in height above the level of the enterostomy, rising with inspiration and falling with expiration. The optimal rate of flow was approximately a litre in one and a half hours. A few seconds' warning of impending vomiting was given by a steady and rapid rise of water in the manometer, and by immediately stopping the flow of the fluid vomiting could be avoided. Atonicity of the intestine was shown by an exaggerated expiratory excursion of the manometer column; under these conditions it was desirable to reduce the rate of flow. If, however, the mean pressure fell permanently, the rate of flow could conveniently be increased. Mucus in the enterostomy tube had a ball-valve-like action; the column began to rise with inspiration in the ordinary way, but was abruptly arrested, falling suddenly again towards the end of expiration. By slightly increasing the fluid pressure for a few moments the tube could be cleared.

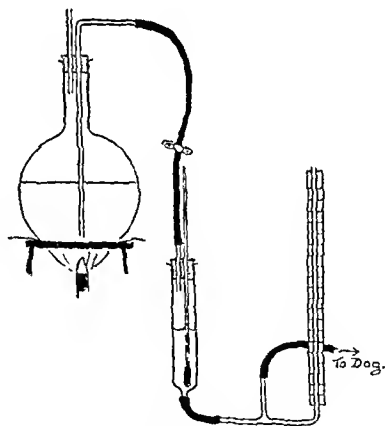


FIG. 351.—Apparatus for continuous feeding by enterostomy.

Daily comparison was made between the intake and output of water in order to ensure a sufficiency being given. It was usually necessary to increase the initial daily intake of approximately 1200 c.c. to 2000 or 3000 c.c. at the end of the first fortnight in order to prevent the development of a negative balance in this respect. The daily intake of foodstuffs and the composition of the diet is shown in *Table I*. (The quantities are those in the daily ration, and are distributed among the three feeds.)

Table I.—COMPOSITION OF FOOD GIVEN TO DOGS 4 AND 6.

Water	1200-3000 c.c.	Butter	1.00 gm.
Sodium chloride ..	10-15 gm.	Marmite	0.50 gm.
Glucose	50-100 gm.	Desiccated bile ..	1.10 gm.
Witte's peptone (trypsin digested)	30-50 gm.	Calcium chloride ..	0.15 gm.
Cystin	0.2 gm.	Magnesium chloride ..	0.05 gm.
Fresh cream	3 c.c.	Potassium chloride ..	0.15 gm.

Bacteriological Examination.—With the object of investigating the question of toxin-formation by *B. welchii*, the following methods were employed.

The stomach contents, drawn off with aseptic precautions at bi-weekly intervals, were used for the preparation of a series of decimal dilutions, and equal amounts of these were added to tubes of Robertson's bullock-heart medium. These tubes were examined after twenty-four hours' incubation, and, if the examination was negative, daily for a week. The growth of *B. welchii* in this medium causes a typical viscous haze and evolution of gas. Identification was completed by examination of the morphological characters of the organism present in the cultures and by the production of stormy clot in milk when cultured anaerobically.

In the earlier experiments an attempt was made to estimate the numbers of *B. welchii* in the stomach contents by making shake cultures in Wilson and Blair's medium, but as the medium frequently failed to give satisfactory results the attempt was abandoned. A rough idea of the numbers of bacteria present was obtained by observing the highest dilution from which *B. welchii* were obtained with the use of bullock-heart medium.

Chemical Examination.—In the blood determinations were made at intervals of three or four days of the carbon-dioxide combining power, the chloride, and the urea nitrogen or non-protein nitrogen. That the first two of these analyses should be made was obviously essential, since one of the chief hypotheses to be tested was that death in high intestinal obstruction was due to alkalosis and depletion of the blood and tissues of chloride. The blood non-protein nitrogen has frequently been reported in the literature as being raised in cases of high intestinal obstruction, and since the kidney function remains normal, it has been suggested that there is excessive breakdown of tissue proteins in this condition—a conclusion which has also been reached from the finding of a high urea excretion. This suggestion we wished to test.

The urine was examined daily. Qualitative tests were made for protein, sugar, and acetone—invariably with negative results—and the daily output of urea and chloride was determined quantitatively. In a few cases the titratable acidity of the urine was determined by titration with sodium hydroxide, using phenolphthalein as indicator.

Before obstruction several analyses were made of the fasting stomach contents. After obstruction the stomach contents, collected as described earlier, were examined qualitatively for blood, bile, and lactic acid, and were subjected to quantitative analysis with respect to free hydrochloric acid (the presence of which was confirmed by Gunzberg's reagent), total acid, and total chlorides.

The various methods of analysis employed, all of which are in daily use in this laboratory, were as follows:—

CO ₂ -combining power	Van Slyke	Blood chloride	.. Modified Volhard
Urea nitrogen ..	Urease	Urine chloride	.. Modified Volhard
Non-protein nitrogen	Micro-Kjeldahl	Gastric chloride	.. Van Slyke

RESULTS.

Appearance, Body Weight, etc.—The dogs used in the earliest experiments of this series are not strictly comparable with the later ones, owing to the presence of intestinal shunts. In *Dog 1* the pancreatic and biliary secretions were shunted so as to enter the intestine below the obstruction, whereas in the later treated animals (which, however, received bile in their food), these secretions entered above the obstruction. *Dog 2*, however, received both bile and pancreatic secretion above the obstruction, and behaved exactly as did *Dog 1*, both as regards time of survival and appearance, and as regards chemical phenomena (Fig. 352). This result not only shows the validity of treating *Dog 1* as a control animal in spite of the difference in surgical procedure, but negatives the suggestion previously mentioned, that the biliary and pancreatic secretions are responsible for the production of a toxin in high intestinal obstruction. In the one case any such toxin was poured in above the obstruction and so remained in the intestine for considerable periods, while in the other it appeared below the obstruction and so escaped as in normal animals. Yet the results were identical in the two cases.

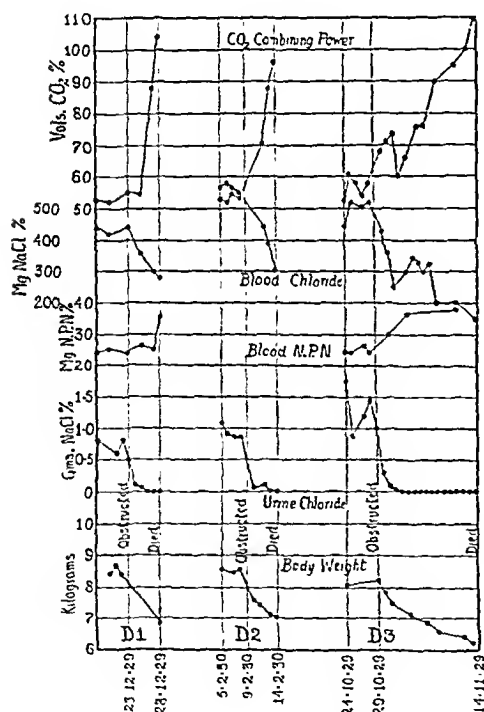


FIG. 352.—Dogs 1, 2, and 3.

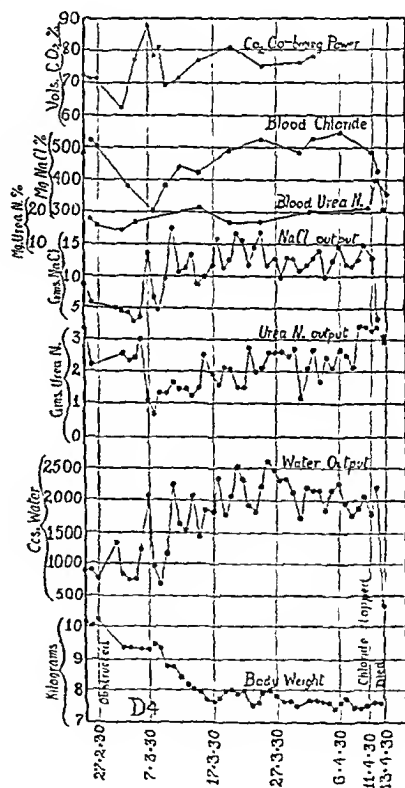


FIG. 353.—Dog 4.

Dog 3, like *Dog 2*, received the biliary and pancreatic secretions above the obstruction, which, however, was lower than in the preceding and most of the later animals, being 22 in. below the pylorus. It, like *Dogs 1* and *2*, presented the classical appearances and chemical changes associated with high intestinal obstruction, the differences between it and the other untreated animals being such as are to be expected merely from the different length of intestine above the obstruction—if the chemical theory is correct. *Dog 3*, in fact, was also a good control animal,

for the greater length of gut available for re-absorption of a possible toxin minimized rather than exaggerated the striking nature of the results of chloride administration in the later animals.

Dog 4 is given as a good example of the results of administration of chlorides, water, and foodstuffs. In contrast with *Dogs 1, 2, and 3*, which rapidly became weak and lethargic, *Dog 4* remained active and mentally alert during the whole six weeks for which treatment was continued. There was also complete absence of the muscular twitching which accompanied the alkalosis of the untreated animals. The body weight, as *Fig 353* shows, fell somewhat rapidly at first, but shortly after institution of peptone feeding the rapid fall was arrested, and thereafter the weight fell only very slowly. When, however, administration of chloride was stopped (although the other ingredients were continued), *Dog 4*, like its predecessors, soon exhibited the characteristic symptoms of lethargy, muscular twitchings, and weakness, and just before death it suffered the typical 'grand mal' convulsions.

Dog 5 (*Fig. 354*), which, after surviving twenty-five days, died with an upper respiratory infection, gave very similar results to *Dog 4*, although it was receiving only chloride and glucose in aqueous solution. Even during the last two or three days of life, when the animal was suffering from the extraneous infection which was the immediate cause of death, there were none of the signs associated with approaching death from high intestinal obstruction. There was greater loss of weight than in *Dog 4*, and the progressive loss continued up to death, since, of course, the animal was receiving no nitrogenous foodstuff. Probably, indeed, the general weakness due to semi-starvation was a predisposing factor to the infection. Nevertheless, the results definitely support those of other workers who have found administration of saline to prolong life in cases of high intestinal obstruction.

Dog 6 (*Fig. 355*), with a simple obstruction two feet below the pylorus, was treated in the same way as was *Dog 4*. This dog lost weight slowly throughout the period of forty-four days during which the treatment was continued, but he remained bright and active, and showed no symptoms whatever. On the forty-fifth day after obstruction, the intake of chloride was stopped, except for the small amount present in the glucose-peptone solution, administration of which was continued. Two and a half days later he was definitely weaker, less alert, and showed the muscular twitchings indicative of an alkalosis. After chemical confirmation of the state of affairs chloride was restored to the diet, and the symptoms disappeared. Three days later, the blood and urine chemistry being normal, the continuity of the intestinal tract was re-established. At operation it was noted that the proximal intestine was markedly dilated, with muscular hypertrophy, to a diameter of $2\frac{1}{2}$ in.—an important point, as indicating the existence of a stagnant collection of fluid affording almost ideal conditions for the growth of bacteria. Thirty-six hours after the operation the dog was given milk by the mouth for the first time, and during the next four days milk and gruel were given in small, frequent meals. At the end of that time minced meat was given, and as no ill effects were produced, full normal feeding was instituted. The weight remained unaltered until full rations were given, when it began to increase, and at the time of writing the dog is in apparently normal health.

Several other dogs, the details of which are not reported here in order to save space, received the saline and foodstuff treatment by administration below the obstruction. They lived for periods varying from twenty-one to thirty-three days, and in no case could death be attributed directly to the obstruction. A progressive

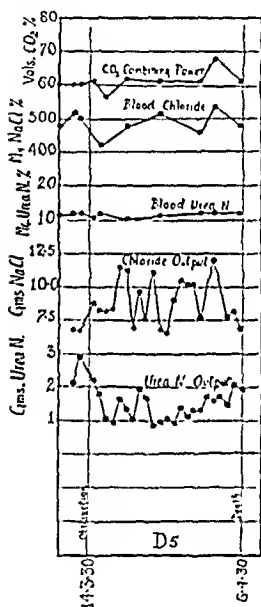


FIG. 354.—*Dog 5.*

loss of weight occurred in every case, which treatment was unable to prevent. The dogs which received peptone and carbohydrate in addition to chloride and water undoubtedly lost weight less rapidly than those which received saline alone. Moreover, both *Dog 4* and *Dog 6* were in a relatively good state of health forty days after obstruction, a result which is better than any previously reported with chloride administration only. It seems, therefore, that if a really adequate diet (for administration by enterostomy) could be evolved, life could be prolonged indefinitely.

Chemical Analyses.—In those animals in which no treatment was attempted the result of the intestinal obstruction was the production of a most characteristic blood-picture, which, it may be noted, was independent of whether the bile and pancreatic juice entered the intestine above or below the obstruction. With a very high obstruction the CO_2 -combining power of the blood began to rise almost at once, though occasionally with a slight delay, due presumably to the acidosis induced by anaesthesia. Simultaneously the blood-chloride began to fall, and these processes continued until, with a severe alkalosis as shown by a CO_2 -combining power of 100 vols. or more, and with the blood-chloride at less than half the normal level, death supervened. During the earlier stage of this process the blood non-protein nitrogen or urea nitrogen remained at or near the normal level, and it was only within a day or two of death—some four to six days after the operation—that there was any marked rise in these blood constituents (*Dogs 1* and *2*, *Fig. 352*). An exactly similar picture was obtained in animals with a somewhat lower obstruction, except that the development was slower (*Dog 3*, *Fig. 352*).

The urine of these animals contained, from the establishment of the obstruction, steadily decreasing amounts of chloride, and from about the fourth day after operation it was practically chloride-free. In spite of this absence or low concentration of chloride, the urine, which was somewhat scanty, had a specific gravity rather higher than normal, and urea, in particular, was present in increased concentration. There was not, however, any increase in the total daily output of urea, but on the other hand there was not, as might have been expected when food was being withheld, a very marked decrease.

The gastric contents never showed any significant diminution in chloride concentration in comparison with the pre-operative fasting contents, and although within a few days of the operation free hydrochloric acid had disappeared (or was present only in very small amounts), the total acid was not greatly decreased. It seemed that there was continued secretion of hydrochloric acid (the tests for lactic acid were negative) at little less than normal concentration, but that this acid was almost completely neutralized by mucins, or, in some cases, by the alkaline duodenal contents. The presence or absence of bile, of course, depended on the site of the

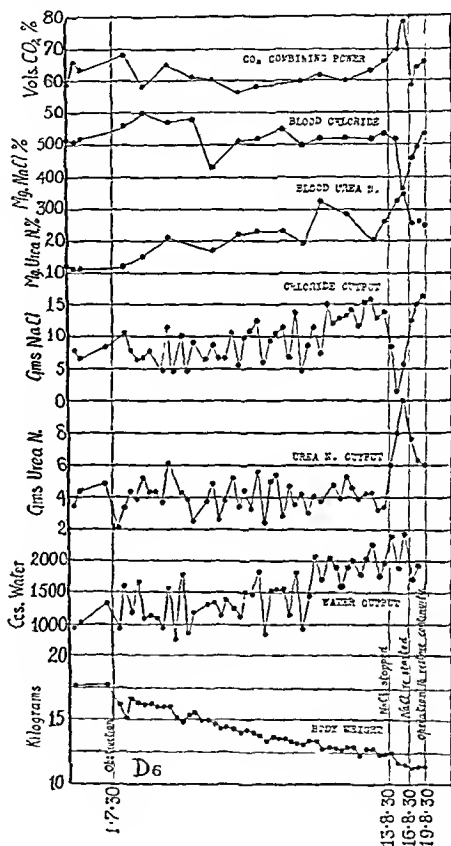


FIG. 355.—*Dog 6*.

obstruction. In those cases in which the bile entered above the obstruction, it was always found in the gastric contents. Lactic acid was never found.

An entirely different picture was presented by those animals which received treatment in the form of saline with or without energy-producing foodstuffs administered below the obstruction. There was no development of alkalosis, and the blood-chloride remained normal. The blood-urea, however, rose slightly in all those animals which were receiving peptone, though not in the one (*Dog 5*) which received chloride alone. Moreover, the increase in the blood-urea became much more marked when the administration of peptone was continued after chloride was withheld (*Dogs 4* and *6*, *Figs. 353, 355*).

As long as the treated dogs received saline, their urine contained chloride, the daily output being of the same order as that obtaining before the establishment of the obstruction. When the animals were in equilibrium, the water intake being so adjusted as to balance the output, the daily volume of urine was as great as that of the pre-operation period, and its specific gravity was within the normal range. The concentration of urea was not increased, and in those animals which received no nitrogenous food the daily output of urea was diminished. The animals which were given amino-acids, either as peptone or as pre-digested protein, showed a proportionate but not excessive output of urea. In the stomach contents of the treated animals the chloride concentration remained at the usual high level, the total acid was in normal concentration, and free hydrochloric acid continued to be present, though certainly it was often in lower concentration than during the control period.

When the administration of chloride to one of these animals kept alive by treatment (*Dog 4*) was stopped, death followed in a short time, with the typical fall in the blood chloride, the terminal rise in the urea nitrogen, and the disappearance of chloride from the urine. Similarly, when chloride was withheld from *Dog 6*, the alkali reserve increased, the blood-chloride fell, the blood-urea rose still further, chloride disappeared from the urine, and the usual clinical symptoms associated with death from high intestinal obstruction made their appearance. In this case, however, the feeding of chloride through the fistula was resumed, and coincidentally the chemical appearances returned to normal and the clinical symptoms vanished.

Bacteriological Results.—The results of the bacteriological examination of the stomach contents are given in tabular form in *Table II*. They call for little comment. No difference is discernible, so far as occurrence of *B. welchii* is concerned, between the treated and the untreated dogs. It would appear, therefore, that the production of *B. welchii* toxin cannot be regarded as the cause of death in high intestinal obstruction. The fact that occasional specimens in each series show complete absence of *B. welchii* is curious. It seems possible that these specimens were collected from a more acid part of the stomach than the others, and this possible explanation is not negated by the fact that the same technique was used throughout. Without some such explanation as this the result seems inexplicable.

Table II.—*B. WELCHII* IN ASPIRATED SAMPLES.*

DOG	PRE-OPERATIVE			POST-OPERATIVE											
1	0	0	No results	5	9	}	Controls								
2															
3	0	0		9											
4	5	3		9	9	9	9	0	9	0	3	0	0		
5	0	0		0	0	0	9	9	9						
6	0	0		0	0	7	0	9	3	0	0	0	9	9	9

*Figures represent greatest decimal dilution in which *B. welchii* was detected.
Specimens taken bi-weekly.

DISCUSSION OF RESULTS.

The chemical results obtained from the untreated control animals are entirely susceptible of the classical explanation based on the chemical theory of death in high intestinal obstruction. The secretion of acid into the stomach and its loss by vomiting leads to an alkalosis. The continued increase in the degree of alkalosis even after the stomach contents have ceased to contain free hydrochloric acid is not a serious difficulty, since, as our results show, there is always a considerable amount of so-called 'combined acid' in the vomitus. This combined acid, of course, represents a net loss of chlorine ion to the blood—with the corresponding sodium ion left behind—since it is merely hydrochloric acid combined with some exceedingly weak base, such as protein. The simultaneous loss of mineral chloride in the vomitus naturally reduces the blood-chloride far below the normal level in spite of the attempted conservation of supplies by the reduction or abolition of the urinary excretion of chloride. The loss in the vomitus is not confined to chloride. The water itself is derived from the body stores, and its permanent withdrawal leads to a growing dehydration with, in consequence, a diminished output of urine.

In these untreated dogs our results show no greater output of urea than is to be expected in early starvation—that is, no excessive breakdown of tissue protein which can be regarded as due to the obstruction rather than to mere deprivation of food. The scantiness of the urine, however, leads to a greatly increased concentration of urea in the urine. It is remarkable that the only increase in the blood-urea occurred in those animals which were receiving treatment, and then only in those whose diet included nitrogenous food. It seems fair to conclude that an increase either in the blood-urea or in the breakdown of tissue protein is not an essential feature of high intestinal obstruction. It is true that a blood-urea increase appeared in the two dogs which, after treatment for some time, were placed on a chloride-free diet, but they differed in two important respects from untreated animals. In the first place, their continued loss of weight showed that they were not able to utilize, even if they were receiving, adequate amounts of food from the artificial dietary. Hence they must, at the time of the chloride deprivation, have been nearer the end of their fat and carbohydrate stores than were the untreated animals. Secondly, they, unlike the untreated animals, continued to receive considerable amounts of water and to excrete large volumes of urine. Either of these factors would account for an additional production of urea, and both may have been concerned in the observed increase. However that may be, it seems that some explanation other than the mere existence of the obstruction is required to explain the phenomenon.

The most important question for discussion—at any rate from a theoretical standpoint—is whether the chemical changes in the body fluids are the cause of death in intestinal obstruction, or whether the cause must be sought in some other factor, such as the production of a toxin, the chemical changes being either secondary or of no account. Naturally, the chemical evidence from untreated cases alone is insufficient to answer the question.

It is not known whether a severe reduction of blood-chloride and consequent alkalosis is of itself capable of causing death, though this may be suspected. Even when it is shown that the administration of saline prevents the blood changes attendant upon intestinal obstruction and at the same time prolongs life, one is not justified in concluding finally that death is ordinarily due to these changes. It is highly probable, but it is not completely proved. It still remains possible that the administration of chloride has altered some other factor of which no cognisance has been taken, and that this factor, which develops only when the blood-chloride is lowered, is the real cause of death. Such a supposition seems unlikely, and, in particular, the continued high concentration of chloride in the gastric contents tends to negative the supposition that lack of chloride aids toxin absorption. Still, the possibility remains, though very considerably weakened when one takes into account the observation that in treated and untreated animals alike there is copious bacterial growth in the stomach contents, and equal possibility in both, therefore, of the production of a toxin.

With equal production of toxin, any difference between treated and untreated animals with respect to the production of a toxæmia must lie in the absorption of the toxin. Were toxæmia the cause of death, however, one would expect the reverse of the actual state of affairs; one would expect that the shorter the intestine above the obstruction, the longer would be the time of survival. For the longer the intestine, the greater the rate of absorption, and so the more rapid the development of the toxæmia. Nor does the difference seem to be due to the absence or presence of bile and pancreatic juice, for in our experiments there was no difference (either in time of survival or in chemical and bacteriological findings) between dogs which received these secretions above the obstruction and the dog which received them below.

On the other hand, the fact that the rate of development of the blood and urine picture parallels the time of survival, and is slower with a low obstruction than a high one, is readily explicable on the 'chemical' hypothesis of the cause of death. With the obstruction at the most rapidly fatal site, near the pylorus, the acid secretion of the stomach is lost with little or no chance of reabsorption. Any alkaline secretion there may be, entering the intestine below the obstruction, can be re-absorbed, but if it is re-absorbed it will aid rather than prevent the development of the alkalosis. The lower the obstruction, the greater the length of small intestine available for the re-absorption of the chloride secreted into the stomach, and therefore the less the permanent loss alike of hydrochloric acid and neutral chloride. Hence the low obstruction leads to a slow loss of chloride and a slow development of alkalosis. Since, however, the process is merely slower than with the higher obstruction and is essentially of the same nature, the terminal picture is the same in the two cases.

Though the chemical evidence alone cannot justify the conclusion that death in intestinal obstruction is due to purely chemical causes and not to a toxæmia, the bacteriological and other evidence makes this conclusion extremely probable. The sum total of the evidence does, to our minds, amount almost to absolute proof, and we consider that in the absence of

fresh evidence the hypothesis that death is due to a toxæmia must be held as disproved.

It has been stated by Cooper⁹ that "no theory as to the cause of death in high intestinal obstruction has given rise to any practical means of preventing death when the condition has once fully established itself." From the experimental results obtained in this work and applied in the case of *Dog 6*, it seems that success in treatment will depend on the following conditions:—

1. Rapid restoration of the body chlorides and water by subcutaneous and intravenous injection of saline. This should be initiated as early as possible, and may be carried on during operation for relief of the obstruction. At least two litres of physiological saline should be given at this stage.

2. Maintenance of the supply of both chloride and water until re-absorption from the alimentary tract has been resumed, as occurs when continuity is restored by operation. It seems, from the experimental results, that as much as ten litres per day of physiological saline or of Ringer's solution may be required at first, the actual amount being regulated by determinations of the blood-chloride. As a rough guide to the level of the blood-chloride the urine may be tested with silver nitrate and nitric acid; the appearance of a precipitate of silver chloride indicates that the blood-chloride is near the normal level.

SUMMARY.

1. Death in untreated cases of high intestinal obstruction is preceded by the progressive development of severe alkalosis, by gross lowering of the blood-chloride, and, at the last, by an increase in the blood-urea.

2. For some days before death the urine is chloride-free and the stomach contents contain no free hydrochloric acid, though their total chloride content is normal.

3. It is immaterial whether the bile and pancreatic juice enter the intestine above or below the obstruction. The time of survival is greater the lower the obstruction.

4. Administration of both chloride and water below the obstruction causes the chemistry of the blood, urine, and stomach contents to remain normal, and also greatly prolongs life. Death ensues only after some four weeks and cannot be attributed directly to the obstruction.

5. With peptone and carbohydrate added to the chloride and water, life can be prolonged for seven weeks or more. Withdrawal of chloride then brings about death, with the usual clinical and chemical symptoms.

6. The occurrence of *B. xelchii* in the stomach contents is the same in treated and untreated animals.

7. It is concluded that death is due not to toxæmia but to the chemical changes following on loss of chloride and water, and is prevented by their supply.

8. Treatment, therefore, consists in supplying chloride and water until continuity of the alimentary canal has been re-established.

9. There is no evidence that excessive breakdown of tissue protein is an essential accompaniment of high intestinal obstruction.

It is a pleasure to thank Professor D. P. D. Wilkie for the interest he has taken in this work and for valuable criticisms, Dr. W. O. Kermack for suggestions as to the composition of the diet, and Messrs. Duncan & Floekhart for the free supply of proteolytic fluid and desiccated ox-bile. One of us (T. C. M.) holds a Grocers' Company Scholarship, and two of us (C. P. S. and D. M. D.) are in receipt of part-time grants from the Medical Research Council. In addition, one of us (C. P. S.) holds a Carnegie Teaching Fellowship. The expenses have been partly defrayed by a grant from the Moray Research Fund of Edinburgh University.

REFERENCES.

- ¹ WILLIAMS, B. W., *Brit. Jour. Surg.*, 1926-7, xiv, 295.
- ² WHIPPLE, G. H., *Jour. Amer. Med. Assoc.*, 1915, lxxv, 476.
- ³ GAMBLE, J. L., and ROSS, S. G., *Jour. Clin. Invest.*, 1925, i, 403.
- ⁴ HADEN, R. L., and ORR, T. G., *Jour. of Exper. Med.*, 1923, xxxviii, 55.
- ⁵ WHITE, J. C., and FENDER, F. A., *Arch. of Surg.*, 1930, xx, 897.
- ⁶ MATSUKURA, S., *Jap. Jour. Med. Sci.*, 1930, ii, 1.
- ⁷ MAURY, J. W. D., *Jour. Amer. Med. Assoc.*, 1910, liv, 5.
- ⁸ WERELIUS, A., *Ibid.*, 1922, lxxix, 535.
- ⁹ COOPER, H. S. F., *Arch. of Surg.*, 1928, xvii, 918.

VICARY'S PREDECESSORS.

BY G. E. GASK, C.M.G., D.S.O.,

PROFESSOR OF SURGERY AT THE UNIVERSITY OF LONDON.

*(Being the Vicary Lecture delivered at the Royal College of Surgeons of England,
on November 6, 1930.)*

THOMAS VICARY, whose name we honour to-day, was Serjeant Surgeon to King Henry the Eighth. This lecture was founded by the Barbers Company to commemorate a man who was Master of their Company and who held a distinguished position in Surgery. His portrait hangs on the wall of this College, his life has been described in happy manner by his biographers, and, though he lived four hundred years ago, our feelings towards him are of intimacy and friendship. Of his predecessors in the office of King's Surgeon, much less is known, and though they must have been men famed in their day, the majority have faded into obscurity. The object of this paper is to rescue them from oblivion, and to put on record the names, at least, of these men who were chosen to attend their sovereigns in peace and in war, and who by their work contributed to make British Surgery what it is.

Unfortunately their biographies cannot be given in great detail, for the lives of medical men in mediæval times have to be drawn up from varied and all-too-seanty sources. There is no book in which the names and services of these men are gathered, but only indirect references scattered through the dusty files.

The source of much of the information gathered for this lecture has been taken from the various Rolls. The first important series of these are the Pipe Rolls, on which were entered particulars of sums of money paid out by the Exchequer. Then there are the very important records known as the Patent and Close Rolls, which form a continuous series from the reign of King John. On these were enrolled copies of the letters-patent, that is, those open and public, and close letters, meaning private letters, which were sent out in the King's name on all sorts of business. These may be described as the equivalent of the duplicate correspondence files of modern government departments. These letters were written on parchment skins, which were sewn together end-to-end and then rolled up. Hence is derived their name of 'Rolls'. These Rolls have been transcribed and printed and so made available for students. They deal with an infinite variety of business, ranging from grave affairs of state to petty details of rewards and punishments to faithful servants or erring subjects.

Among this mass of material one finds the scanty notices of our early predecessors. These notices, generally brief, commonly refer to some grant of land or money conferred on them as wages or rewards for services rendered, and it is for this reason that their names have been preserved.

From these and other similar sources a continuous sequence of medical officers of the reigning sovereigns can be traced back as far as the time of Edward the Confessor. Sometimes merely the names are recorded, but here and there a few details emerge, trifling details for the most part, but enough to throw a little light on the distant past.

In the eleventh century, in which our first record comes, medicine was largely, if not entirely, the prerogative of the Church. Learning was confined to the monasteries, and the aspiring cleric learnt medicine along with grammar and philosophy as one of the humanities. Thus, Abbot John of Cella, of St. Albans, was eulogized as "a Priscian in Grammar, an Ovid in Verse, and a Galen in Physic". Nor did these early physicians confine themselves to the practice of medicine. They were first and foremost clerics, and for their medical services to their sovereigns they were commonly paid by gifts of livings.

The first of Vicary's predecessors to be recorded here is BALDWIN, the Abbot of Bury St. Edmunds, who was physician to Edward the Confessor. He was a cultured man, and described as "gretly expert in craft of Medycyn".

Following him came NIGEL, whose name figures in Domesday Book as the recipient of various gifts of land. He was physician to William the Conqueror. There is no record of his presence at the Battle of Hastings. The Bayeux Tapestry gives us some idea of the treatment of the fallen (*Figs. 356-359*).

William Rufus had as his physician JOHN DE VILLULA, a learned divine, coming, it is believed, from Tours. He became Bishop of Bath, where he founded a monastery which became a seat of learning.

During the reign of Henry the First, the Pipe Roll mentions the names of GILBERT DE FALAISE as 'Medicus'; of GRIMBOID who lived in the Royal Court; and of CLARIMBOID who was physician and chaplain.

In the reign of Stephen appear the two shadowy names of ERNOLF and IWYN. Little is known of these beyond the fact that their names appear as witnesses to certain documents.

The Pipe Roll of the reign of Henry the Second includes the names of a number of medical men, such as JOHANNES medicus and RUDELFUS medicus, and there is the faint echo of one tragedy in a terse record that MASTER RALPH DE BELLOMONT, King's physician, was drowned in the Channel in 1170.

Norman Moore described MAGISTER RAMULPHUS BESACE as physician to Richard the First in Palestine, and George Parker, in his *History of Surgery*, quotes a reference from the Pipe Roll to the effect that MASTER JOHN OF BRIDPORT, medicus regis, should have a suit of clothes when he went to Normandy.

The reign of King John is not productive of much information, though there is one note in the Close Rolls ordering that the necessaries for three horses should be provided for MASTER R. FUKET,¹ the King's physician, which shows that even in these early days some arrangements were made for the provision of clothes and necessaries for the physicians attached to the King, but it is not till the reign of Edward the Second that we find an orderly account of their rights and duties set out in the Household Ordinances.

It will be noticed that up till now there has been no mention of surgeons,

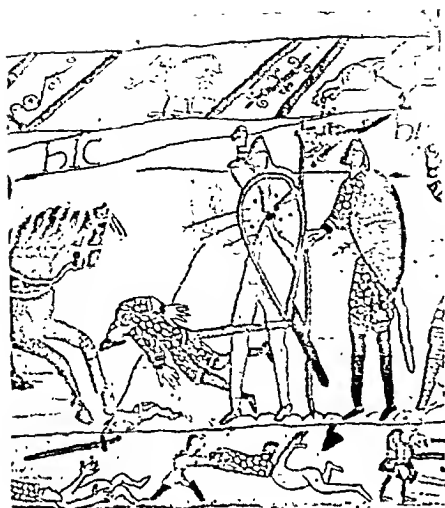


FIG. 356.—The Battle of Hastings from the Bayeux Tapestry. The scene depicts the day going against the Saxons. The lower border shows how the fallen were stripped of their armour.

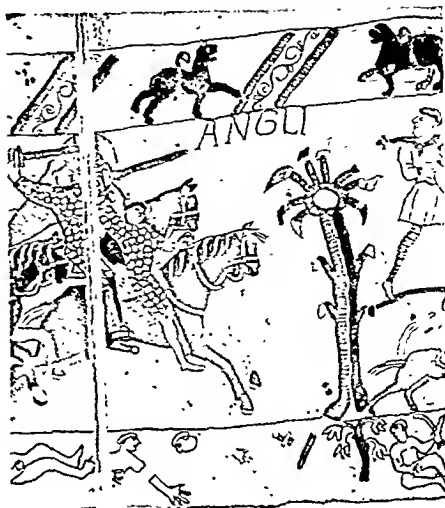


FIG. 357.—The last scene from the Bayeux Tapestry. The Saxons fly pursued by the victorious Norman horsemen.

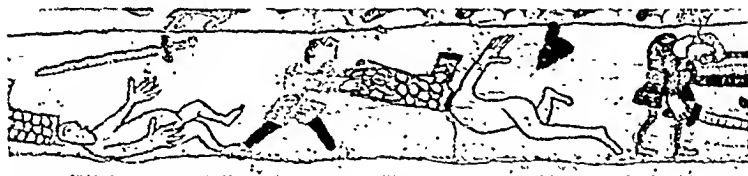


FIG. 358.—Enlargement of the lower border of Fig. 356. The liveliness of the picture indicates that the men were stripped while still alive.

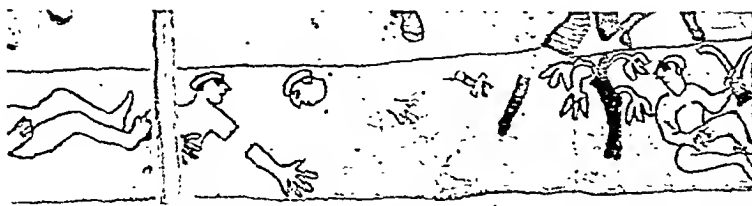


FIG. 359.—A figure from the lower border of Fig. 357. This shows a wounded man, stripped of his armour, sitting up and holding a piece of mandragon, the well-known narcotic herb used in the Middle Ages.

but only of physicians. It has to be remembered that in these early days doctors were physicians and surgeons too, for at that time medicine had not thrown away her right hand of surgery. The reasons why surgery was divorced from medicine—a fact we still deplore—and the date at which the separation took place, are still obscure, though the subject has been hotly disputed. Suffice it here to say that the separation did take place, and probably sometime during the twelfth century.

It is for this reason that we do not find records of a separate appointment as a surgeon to the early kings. In fact it is not till 1233, in the reign of Henry the Third, that the scanty records yield a notice of a medical man having the title of Surgeon to the King. The first of these surgeons, as far as I have been able to discover, was Master William.

MASTER WILLIAM.—In the Calendar of Patent Rolls given at Woodstock on 12th May, 1233, in the 17th year of the reign of Henry the Third, appears the first notice of an appointment of a surgeon to a king.²

Presentation to Master William, the King's Surgeon to the Church of Honkerinton, in the same diocese (Lincoln) in the King's gift by reason of the land of Thomas, son of Walter, being in his hands.

From this scanty notice it can be inferred that William was an educated man and of standing in his profession, seeing that he held the title of Master; also that he was a cleric, and that he was being rewarded for his services by presentation to a living. More than that one cannot say, for with this solitary reference he appears and disappears in the pages of history.

Almost equally obscure is the second surgeon of whom there is record, namely, MASTER DAVID. In 1241 there was a mandate from King Henry

the Third to the Justiciary of Ireland to provide, as soon as opportunity offer, Master David, the surgeon, with 100 shillings of land in escheats or wards.³ Again, two years later, in 1243, an order was given to the Justiciary of Ireland to give possession (*seisinam*) to Master David, the surgeon, of certain property of John of Kaerdiff.⁴ Master David must have died about this time, and in



FIG. 360.—Henry III sailing home from Gascony, 1243. Drawn by Matthew Paris. (From Green's "*Short History of the English People*", by kind permission of Messrs. Macmillan & Co. Ltd.)

not too flourishing conditions, because a few years later, in 1248, King Henry made an order, bestowing on Alice, the widow of Master David, formerly the King's surgeon, for her maintenance during her life, those three obols (*obolos*) which Brother Nicholas, the Westminster recluse, used to have through the charity of the King.⁵

After the death of Master David it would appear that his successor was MASTER HENRY DE SAXEBY. At any rate we hear of him first in 1251, and he is notable among other things for being the first surgeon to carry the title

of Serjeant Surgeon. It is difficult to be sure of the exact meaning of the title in these times. The first reference to it is as follows:—

Grant to the King's serjeant, Master Henry, the King's surgeon, King's clerk of £10 at the Exchequer etc.⁶

The reading of serjeant in the Latin in which the Rolls were written is 'serviens', that is to say 'servant'. The King had many such serjeants attached to his person; thus there was a serjeant of the napery, a serjeant-at-arms, and a serjeant of the pavilion. It seems probable that at this time the description meant that such a one was attached to the King's person and received grants of land or livings in lieu of wages. Later it appears to have meant that, when more than one surgeon was attached to the monarch, the senior of them was termed 'Serjeant Surgeon' and was responsible to the King for the surgery of the Court and for providing and keeping the necessary drugs. Be this as it may, Master Henry de Saxeby seems to be the first of the long line of Serjeant Surgeons which came down through Thomas Vicary and is continued to this day in the person of Sir Hugh Rigby, K.C.V.O.

Now for the first time we hear what was the salary of the King's surgeon. This was laid down in the case of Master Henry and was £10 per annum, which he was to receive at the Exchequer until the King could provide for him in land to that yearly value to hold for him and his heirs.⁷ This promise apparently was made good, for about the same time appears a notice that Master Henry had served the King well, and the King, desiring his promotion, commanded the Justiciary of Ireland to provide 10 librates of land.⁸

There is one little incident of Master Henry de Saxeby's life that has been preserved, and is of interest as showing the manner in which working men were treated in those days. In 1251 a wall in Westminster Hall collapsed, and in its fall injured seven men. Master Henry was detailed by the King to look after these men, and the King paid also for their food, attendance, and necessities for their cure.⁹

The most remarkable surgeon of the period was MASTER THOMAS DE WESHAM, who must have been a man of considerable attainments and character, for he held the position of surgeon to the King for some twenty years. He has been well known for many years to coin collectors, but little has been said of him as Serjeant Surgeon to Henry the Third.

Of his birth and education unfortunately little is known, though it may be inferred that he was a layman and not a cleric, and that from his name he was probably an Englishman. He may have taken his name from the little town of Wescham in Lincolnshire, and he was at any rate a contemporary of the famous Roger of Wescham (d. 1257), who was Lecturer on Divinity at Oxford, and later Bishop of Lichfield. It is pleasant to think of the possibility of these two men being brothers, one an eminent divine, and the other a distinguished surgeon.

We first hear of Master Thomas of Wescham in 1253, when he is cited as amongst those accompanying the King in one of his expeditions to Gascony.¹⁰ It seems probable that he went in the capacity of body surgeon to the King, and that he had as his companion MASTER PATRICK DE CARLIOLO, the physician. The accompanying picture (*Fig. 360*) is Matthew Paris's idea of how they came home.

After the return from France several gifts of land were bestowed on Master Thomas. In 1255 he was given a house in Colechester which had belonged to Isaac, the Jew,¹¹ and the following year was granted for life the bailiwick of the Forest of Cannock.¹² In succeeding years he received other grants, but one is of special interest and unusual. In 1260 the King granted Master Thomas a die in London, that is, he was authorized to mint coins, and presumably there was a certain amount of profit attached to the right.¹³



FIG. 361.—Anron of Colechester, Forest Roll, Public Record Office, (From Green's "Short History of the English People", by kind permission of Messrs. Macmillan & Co. Ltd.)

Mr. L. A. Lawrence, a Fellow of this College, possesses a coin minted by Wescham, and by his kindness it is reproduced here (Fig. 362). The reading on the coin is: 'Henricus Rex III' on the obverse, and 'Thomas on Lund' on the reverse side.

For twenty years after the gift of the die, Master Thomas of Wescham and his wife, Sarah, lived in the Parish of St. Michael in Bassieshaw.¹⁴ It is pleasant to think of this honoured surgeon spending his declining years in peace and quiet in the City of London.

It would seem that Wescham was succeeded in the late years of Henry the Third's life by another surgeon, namely, MASTER SIMON DE PRESTON. There is little to record about him beyond the fact that in 1271 he was exempted for life from being put on assizes or from being made sheriff against his will.¹⁵ Thus early in mediæval times medical men were granted privileges similar to those which are enjoyed by members of this College to this very day.



FIG. 362.—A coin minted by Thomas de Wescham, Surgeon to Henry III. (In the possession of L. A. Lawrence, Esq., F.R.C.S.)

The long reign of Henry the Third is thus notable for the appearance of surgeons, as apart from physicians, and also for a great spurt in the study of medicine. All over the continent of Europe hospitals were springing up, and the same was happening in England. Henry was a Frenchman, and his Queen, Eleanor of Provence, was a Frenchwoman. People complained that he filled the chief posts of this island with a horde of hungry relatives. Certainly many French doctors came over, and no doubt brought with them learning from the French Universities which was of inestimable value to us.

Not only did Henry the Third have his own medical and surgical attendants, but he provided a medical establishment for his Queen, Eleanor of Provence, and also for his son, Edward, later Edward the First, and probably most of these were Frenchmen.

Edward the First was cast in a different mould from his father. Henry, and during his long minority his character was so formed that by the time he became King he had acquired a reputation as a statesman, organizer, and general second to none in the kingdom.

It is during this reign that we get the first glimpse of an ordered household, with list of the duties and emoluments appending to the various offices, and, according to Withington, the germ of an organized army medical service. Indeed, Garrison gives Edward the credit of making the first attempt at the formation of a military medical service in the Middle Ages, apart from the Byzantine Empire.

Edward the First had two notable surgeons, MASTER SIMON DE BEAUVEYS (de Belvaec), and his son, PHILIP. One may suspect that Master Simon was a Frenchman, taking his name from the town of Beauvais in France, but there is nothing to tell about his early life or where he got his training. His son, Philip, was probably brought up in England and spent his early life about the Court, entering the King's service at an early age, for in 1279 he is described as King's yeoman and receives a grant of 200 marks to get married.¹⁶

Many grants of land were made to these two, which it would be wearisome to relate. The share, however, which Master Philip took in Edward the First's campaigns against Scotland is of interest as illustrating the manner in which war was then waged.

Of the five campaigns against Scotland, those of 1298 and 1300 are of special interest, because in the records of these are the earliest indications of anything like an organized army medical service.

In order to make the matter clear it is necessary to give a short account of these expeditions.

In 1298 Edward the First led an expedition against the Scotch who were ravaging the North of England. The army consisted of cavalry and infantry, led in its several companies by bannerets, and was divided into four battalions, the third of which was commanded by the King himself. Besides bannerets the host included simple knights and valets, who appear to have been in rank equal to esquires. It was the practice to appraise or value the horses of knights and others at the time when they joined the army, and to pay for such of them as were killed or lost in the King's service; and it is from these lists that some of our information about the doctors is derived.

The English forces were not opposed until they came to Falkirk, where they found Wallace and the Scots drawn up in battle array behind a morass, and the Battle of Falkirk was fought on July 22, 1298. It was summer time, and the night before the battle the English troops lay on their arms, and the King laid his long shanks on the bare ground beside them. During the night a sudden alarm startled the horses, and the King's charger trampled on him and broke two of his ribs. This accident, however, did not prevent the King leading his troops to battle and victory. The Scots were defeated with very

great loss, while the English lost only a few men. After the battle was over, King Edward, feeling no doubt in pain from his injury, repaired, according to Rishanger, to Stirling, and lodged in the Convent of the Black Friars to convalesce.¹⁸ According to Comrie, however, King Edward went for his convalescence, not to Stirling, but to the Priory or Hospital of Torphichen, situated a few miles away, and which still stands to this day.¹⁹

It seems more likely that he should have gone here, because the Priory belonged to the Knights Hospitallers of the Order of St. John of Jerusalem, probably an off-shoot of the well-known Priory in Clerkenwell; and moreover the head of the Hospital was an Englishman, Alexander de Welles, who was killed at Falkirk fighting on the English side.

There were a certain number of medical men included in the lists of those present with the King. First there was a physician, MASTER JOHN DE KENLEYE, who is mentioned as having letters of protection on account of being about to set out to Scotland in obedience to the command of the King (*in obsequium regis*). In the Roll of the horses of the men attached to the royal household, Master John de Kenleye is also noted as having a horse, dappled or iron-spotted, valued at 12 pounds; also that he had a valet (*valettus*) who had a horse, iron-grey, valued at 6 marks. The valet's name was William le Mareschal.

It is not clear as to the meaning of the word *valettus*. It may mean that the physician was accompanied by one of his own profession, or it may mean that he was a soldier detailed to protect and serve his master.

The only surgeon who is described as being present is MASTER PETER (*Cirurgicus Regis*). He had a horse with a star on his forehead valued at 16 marks, and his valet, John le Mareschal, also had a horse valued at 16 marks.

It is uncertain whether Philip de Beauveys was present in this campaign: probably he was, and possibly it was he who diagnosed the fracture of the King's ribs.

As far as can be ascertained no arrangements were made for the care of the sick and wounded, and there are no details about wounded men.

The Scotch Campaign of 1300.—In April, 1300, Edward conducted a successful campaign against the Scotch. Fortunately for us the Wardrobe account of this, the 28th year of the reign of Edward the First, has been preserved, giving detailed accounts of disbursements. This includes many interesting details concerning the medical men who were present, their pay, the clothing they received, and the horses they had.²⁰

The actual facts, as far as they can be ascertained, are that the King had in his train, his physician, Master John de Kenleye, with two servants, or perhaps juniors (*valetti*); also his surgeon, Master Philip de Beauveys, and two assistants (*socii*); and in addition there was Master Peter, who was noted as being present in the campaign of 1298. The King's physician and surgeon each received a knight's pay, two shillings a day, and the others who ranked as esquires half that sum. In addition to their pay, the King's physician and surgeon each received four marks for clothes for summer wear and four marks for clothes for winter wear, and while they were with the Court they received their board and lodging free.

Master Peter, who seems to have been a junior officer, only received one mark for clothes instead of four, as his seniors did.

It is open to argument whether the four men described as '*valetti*' and '*socii*' were really medical men, as has been supposed by Withington. They may have been, but there is nothing to substantiate the theory.

In support, however, of the theory of an organized medical service, it should be stated that Master Philip, the surgeon, was allowed the sum of 40 shillings annually for the purpose of supplying diverse medical stores for the use of members of the household who might be injured, and Master Peter was allowed half that sum. Master Philip must have been hard worked during this campaign or have had bad luck, for he lost three horses in the service of the King, for which loss he was compensated at the rate of 40 shillings a horse. The records do not say whether these horses were killed in action or died of disease.

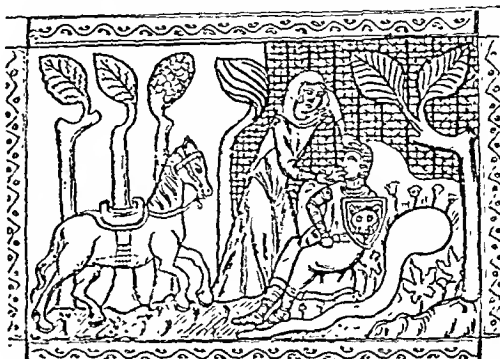


FIG. 363.—The chataine administering mandragon to a wounded knight. (Published in "*Chirurgiens et Blessés*", by Cabanes.)



FIG. 364.—A wounded knight transported by a comrade to a monastery. (Published in "*Chirurgiens et Blessés*", by Cabanes.)

It is not quite clear whether Master Kenleye, the King's physician, was present during the whole of the campaign or not. For part of the time he was sent off by the King to Caversham to attend the King's daughter, the Countess of Gloucester, who was ill. During the time he was away from the Court, Master Kenleye received one shilling a day.

A careful search has been made to discover what arrangements were made for the care of the sick and wounded in these campaigns, but practically nothing has come to light. There is evidence that some sort of consideration was given disabled soldiers, for it is recorded that a certain Harvey de Cornubia, a valet of the King's napery, who was wounded by the Scots in Galway, was given his

expenses to pay for his return to England and for his necessary medicines. But as for the immediate treatment of the wounded, nothing seems to be known. Figs. 363, 364, taken from the French romance of *Lancelot du*

Lac, would seem to illustrate the sort of thing that happened. Here is a wounded knight tended by a fair lady, an early prototype of a nursing sister, who by her association with mandragon obviously was accredited with a knowledge of drugs and salves. The second picture shows a wounded knight taken by a comrade to a monastery to be cared for by the hospitable monks.

Some such methods as these we may suppose were used, for there is no hint of organization for the care of the wounded. We have to wait for nearly two hundred years, till the time of Queen Isabella of Spain, for the establishment of Camp Hospitals and an Ambulance system.

Edward the First died in 1307, while leading yet another expedition against Scotland, but his death only arrested for a moment the advance of his army. Edward the Second, however, had neither his father's skill as a general nor his gift to govern. As is well known, the Battle of Bannockburn, in 1314, resulted in the total defeat of the English, the King narrowly escaping with his life. Very few details of this disastrous battle have been preserved, and we know nothing of the medical men who may have been present or of any of the medical arrangements. After this campaign a number of inconclusive expeditions resulted, in 1323, in a thirteen years' truce between the two countries.

One of these expeditions, however, that of 1322, deserves a special mention, not on account of its military importance, but because, among the few details which have been preserved, there is a record of the medical stores which were sent up from London to Newcastle by water for the use of the troops by MASTER STEPHEN of Paris, the King's surgeon.

This document, the earliest of its kind, proves beyond doubt that there was at this time some sort of organization to provide for the medical needs of the army, and that some person or body of men were entrusted with the preparation and despatch of the material. In this instance it looks as if the person whose duty it was, was the King's surgeon, Master Stephen of Paris. An examination of the document shows that the drugs were packed in two great 'paniers', which cost 6d. apiece, and were despatched from London to Newcastle by ship, and thence to Edinburgh by water, and from there back again to Hayleland. The total cost of the goods, including carriage from London, was £91 3s. 9d. It is interesting to note that the term 'panier' still survives in our army, and that it is in them that the medical stores of the Field Ambulances are carried to this day.²¹

The stores themselves, called "drogneries and implastra", are specified minutely, and include 3 lb. of oxeroerosin; 3 lb. of dyataroseos; 6 lb. of apostolicon; 2 lb. of Saungny's Draconis, and various other forms of ointment.

Of further details of the King's surgeon, Master Stephen of Paris, we have none. Beyond the bare mention that he was the surgeon, and that MASTER ISAMBERT was the physician, the records are silent.

Though so little is known about the medical men of this reign, we have some very important details about the offices and emoluments of the King's physician and surgeon. These are included in the Household Ordinances of Edward the Second, and there we find them set out clearly and definitely for the first time.²² Whether they were so set out and defined by Edward

the Second is not certain. One suspects that they date back to earlier reigns, though there is no direct evidence as to when they were first drawn up.

The Ordinances were written in old French, and the following is a modernized version :—

A Chirurgien.—The surgeon shall have his diet every day in the hall, if he is not hindered by some business certified before the Steward and Treasurer. And then he shall have his livery as a knight of the household, whether he be well or ill, that is to say, two darres of bread, one piteher of wine, two messes de gros from the kitchen, and one mess of roast. And shall take every day for his chamber, one piteher of wine, three candles, one tontis, litter all the year, and fuel for dinner time, of the usher of the hall. He shall have 12 pence a day wages until he be advanced by the King and two robes yearly in cloth, or 8 marks in money. For things medicinal he shall have forty shillings by the year.

There was a charge for the physician similar in every way, except that the physician was allowed three darres of bread instead of two, and he was allowed wages for three servants.

It will be noticed that the surgeon was allowed forty shillings for the provision of medical stores, and that this was the same sum as was allowed Master Philip de Beauveys for the same purpose in the reign of Edward the First. It looks as if it was the duty of the King's surgeon to provide medical stores for the use of the members of the royal household. The accompanying pictures are illustrative of approximately contemporary customs (*Figs. 365-367*).



FIG. 365.—An apothecary's shop. Thirteenth century. (From the "*Treatise of Surgery*", by Roger of Parma, Sloane, 1977.)



FIG. 366.—The dentist. Fourteenth century. (Roy. MS. 6 E.11.)



FIG. 367.—Bleeding. (From the "*Luttrell Psalter*" f. 61.)

The reign of Edward the Third rings with stories of tossing spears, of gleaming armour, and blazoned shields, stories Froissart loved to chronicle. A glamour still clings round the name of Crécy, though nigh six hundred years have passed.

The battlefield of Crécy remains to this very day very much as it was in 1347. It is easy to mark the position taken up by the small British Expeditionary Force—"that contemptible little army"—and the slope of ground where they withstood the chivalry of France, and the very site of the windmill under which the King stood watching the Black Prince win his spurs.

Who were the doctors at this battle, and what position did they occupy during the fight? Were they with the little group which stood round the King under the old windmill, or were they with the carts which we know were parked not far away in the rear? Their number and their position must be matter for conjecture only, because the records are lamentably lacking in all such details.

It is known that the King's surgeon at that time was MASTER ROGER DE HERTON, and though it is not definitely stated that he was present at the battle, it is probable that he was; and it is most likely that he had at his command medical stores packed in the service paniers which have been mentioned as being provided in the reign of Edward the Second.

At the time of the Battle of Crécy, Master Roger must have been a man of mature years, for he had then been some twenty years in the King's service. The first reference to him occurs in 1328, in the second year of the reign of Edward the Third.²³ He was then described as the King's surgeon, and was granted at the request of Edmund, Earl of Kent, custody of the town of Penmanthlu in Merionethshire. In succeeding years he received a number of grants of land and otherwise, and in one of these grants it is specifically stated that it was in consideration of his services in staying constantly by the King's side. We know also that he received as a gift a house close to the gate of the Palace of Westminster, where he lived with his wife, Isabel, and his two daughters, Alice and Isobel, who were aged 19 and 18 respectively, when he died on May 13, 1349, two years after the Battle of Crécy. The exact date of his death has been preserved on account of a dispute which arose later as to the ownership of this house.²⁴

Whether Master Roger took part in the Battle of Crécy must remain in doubt. It seems clear that he intended to partake in the expedition, for in the French Roll, dated May 15, 1346,²⁵ is preserved a writ to the Bailiff of St. Albans: "to exonerate the King's beloved Magister, Roger de Heyton, the King's surgeon, who was about to set out on the King's service, from any contribution to find armed men on account of his land and tenements in that town."

One cannot refrain from making mention here of the physician who was contemporary with Master Roger, namely, MASTER JORDAN OF CANTERBURY, the King's physician, who certainly was present at Crécy. It is recorded of him that he received as wages of war 109 shillings for a period of three years and a half and 56 days. Master Jordan died about 1361, after some thirty-five years' service with the King.²⁶

Search the records as one will, no mention is made as to the method of picking up or treating the wounded, except a brief reference by Joshua Barnes,²⁷ in which he quotes Giovanni Villani in his description of the events following the battle :—

and so the High Mass of the Holy Spirit was solemnly sung and the Victory acknowledged to the Author thereof. Moreover for the sake of the dead the Pious King caused the place of the Battle to be consecrated for the decent sepulture of his enemies and friends : and that the wounded being removed from among the dead, should be dressed ; to the meaner sort of whom he afterward gave money and sent away.

Who dressed the wounded, and how they were taken away, we do not know. It is stated that there were a certain number of surgeons attached to the Welsh troops, and they may have done the work. As regards transport, we know also that Edward had a certain number of carts which were parked at the rear during the battle, and we may suppose that any wounded that were carried away were taken in these carts. This was the universal way of transporting the wounded, even in the time of the wars of Marlborough and up to the Napoleonic era.

That there was the germ of a mechanism for invaliding soldiers no longer fit for active service, and some sort of pension scheme, is shown by the following record. A writ of protection was given to the Sheriff of the County of Northampton, in favour of Sir Nicholas de Burnaby, who had served in the retinue of Edward, Prince of Wales, and had been severely wounded in the foot by a quarrel. There is also a similar writ in favour of Sir Peter Malore, who had appeared personally in chancery and who was manifestly so maimed that he could no longer give aid in warlike actions ; and another writ to the Mayor of Cambridge, stating that William Brice had returned to England grievously injured, and by an inspection of his wound in chancery it was evident that he could no longer serve usefully.

Though our government records do not throw any light on the method of treating the wounded, there is an illuminating passage in Froissart which is probably a correct picture of the times.

It concerns the famous knight, Sir John Audley, who fought valiantly at the Battle of Poitiers. He was wounded in many places, and when overcome by them, he was led or carried out of the press by his four squires who laid him under a hedge, disarmed him as gently as they could, in order to examine his wounds, dress them, and sew up the most dangerous.

Who taught these squires how to bandage and treat wounds ? I suspect that the chatelaines—the wives of the knight—did, and that part of the duty of the squire was to carry dressings and salves.

The last of the surgeons to King Edward the Third was MASTER ADAM ROTTS, who was first mentioned in this capacity in 1359. He was granted a property in the Poultry, where he seems to have lived and died in 1370. His will has been preserved.²⁹ From it we learn that he had a wife, named Juliana, and a brother, William. To Sir William Stodeley he left a girdle with pouch and knife, given him by the Duke of Lancaster, and among other bequests he gave some house property to the Priory of St. Bartholomew. From this I would like to assume that he was a good Bart.'s man.

having received his training in the old Priory, but as regards this there is no information.

It would not be right to leave the reign of Edward the Third without a mention of the great English surgeon, JOHN OF ARDERNE, who has given us our first book on Surgery, and who no doubt knew the men we have been describing.

Richard the Second had three or four surgeons. It is pleasing to be able to record a kindly action of this rather poor King towards one of them, namely, JOHN LECHE. We hear of this man first in 1382, when he is described as King's surgeon. Then in the following year appears this record:—

Grant, for life, to the King's surgeon, John Leche, whose sight is so weak that he can no longer labour in his office, of the wages and robes yearly as an esquire in the king's household, whether he is present or absent therefrom, as heretofore received by him from the keeper of the wardrobe.

As late as 1391 he is still described as the King's surgeon, and is given a grant of land and is to continue to have his robes or 40 shillings in lieu thereof.³⁰

At least two other surgeons served Richard the Second, namely JOHN SALESBURY and WILLIAM BRADWARDYN. Of the former, John Salesbury,

there is just a bare record in which he is described as surgeon, the King's serjeant (*seriens noster*), and that he was granted 40 shillings for life at the exchequer. Towards the close of the reign, it would seem that the King's surgeon was William Bradwardyn, who had the distinction of serving three sovereigns, Richard the Second, Henry the Fourth, and Henry the Fifth. He will be referred to again later.

Henry the Fourth, the son of John of Gaunt, who deposed Richard the Second, and became King

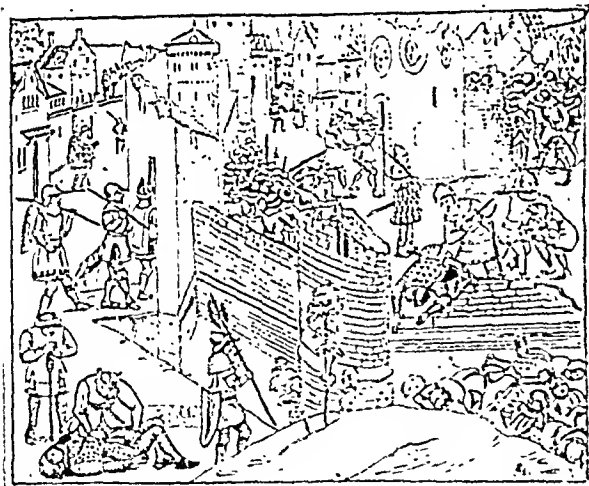


FIG. 368.—A French surgeon operating on the field of battle. (Published in "*Chirurgiens et Blessés*", by Cabanes.)

of England, seems to have brought with him from France some French physicians, whom we may presume he came to know during his term of exile in that country. Their names, at any rate, sound foreign: LOUIS RECOUCHEZ or RECOUCHE, PÉTEN DE ALBOCASSE, and DAVID DE NICARILLIS. The first of these, Louis Reconche, was certainly a foreigner, because he received grant of denisation, or, as we should say to-day, papers of naturalization.³¹

Of the surgeons of Henry the Fourth we have but little to recount. There is one record, in 1410, of a grant for life to the King's servant, THOMAS

MORSTEDE, surgeon, of 40 pounds yearly.³² This is no doubt the Thomas Morstede who is so well known as surgeon to Henry the Fifth (*Fig. 369*).

It is to be noted, too, that the surgeon's salary now is 40 pounds per annum, as against the 10 pounds granted to Master William in the reign of Henry the Third.

The other name which occurs on several occasions in the records of this reign is that of William Bradwardyn, or Bredewardyn. These records refer to him as esquire, surgeon, and serjeant-at-arms. It seems unlikely that there were two men of the same name, so it seems fair to assume that the two surgeons of Henry the Fourth were Thomas Morstede and William Bradwardyn, and that when Harry of Monmouth put on his father's crown and became King of England, he took over his two surgeons as well.



FIG. 369.—An imaginary picture by A. Forestier showing the departure for France of Morstede and a number of pressed surgeons. (*From the Collection of the Wellcome Historical Medical Museum.*)

Henry the Fifth began to reign on March 21, 1413, and he won the Battle of Agincourt on October 25, 1415. The details of the arrangements of the Expeditionary Force that he led are of considerable interest, for they concern the lives of the two notable surgeons—Thomas Morstede and William Bradwardyn. Preparations for the expedition were started a year ahead, and soldiers were enlisted under contract or 'indenture'. In this way, 2500 men-at-arms and 8000 archers were enrolled, and in addition numbers of non-combatants, such as carpenters, smiths, miners and gunners, armourers, yeomen of the pavilions, saddlers, physicians, surgeons, and chaplains.

The chief surgeon to the force was Thomas Morstede of London, and the indenture made with him is still preserved. He was to be ready to

attend a muster in May, 1415: he was to bring with him fifteen persons, of whom three should be archers and the others men of his own craft. His wages for the whole year were to be 40 'mares' for himself and 20 'mares' each for the other fifteen persons. The wages were to be paid in advance for the first quarter. It appears that the duty of organizing this fell to the service of the King's surgeon, who became, as we would style it to-day, Director General of the Army Medical Service.

In May, 1415, Morstede applied for money to purchase medical stores, as well as one cart and two horses to carry them.³³ Here is the germ of a medical service. To our modern ideas, the provision of one cart and two horses seems ridiculously inadequate to carry all the medical stores for an army preparing for a year's campaign. One may recall, however, that as late as 1808, at the beginning of the Peninsular War, only a little over one hundred years ago, when Sir Arthur Wellesley landed in Mondego Bay, the whole of the medical stores for the whole army was loaded into two carts drawn by bullocks. In assessing this campaign, therefore, one may conclude that it was the most carefully planned and the best organized and equipped expeditionary force that had ever sailed from this island. It throws a light, too, on the character of Henry the Fifth: he was not a hare-brained, devil-may-care adventurer, ready to risk his crown and kingdom on a mad expedition, but a rare general who took pains to leave as little as possible to chance.

The other surgeon who joined the expedition was William Bradwardyn. According to Sir H. Nicholas, he was in the retinue of the King, and had with him nine other surgeons.

The Physician to Henry the Fifth was named NICOL COLNET, and an indenture corresponding almost exactly with that of Thomas Morstede was made with him, and his wages were the same; but he was named alone and no other physician accompanied him.

There is not time here to follow the fortunes of the men engaged in this audacious but successful expedition. How, at the Siege of Harfleur, they had to deal with an outbreak of dysentery, in which 2000 men died and 5000 more had to be invalided home. Nor can we depict the hardships they endured in their march up the valley of the Somme, past Abbéville, Pont St. Rémy, Hangest, Corbie, Boves, Nesle, and Harbonnières. It was at Harbonnières that one of the soldiers, Bardolph, according to Shakespeare, stole a copper-gilt pyx from the church. The names of these towns are well known to many, and they are hallowed for all time by the graves of thousands of British soldiers who fell in the Great War.

Tired and lean from long marching, but with stout hearts, this little band of brothers met the French force on St. Crispin's Day, 1415, at Agincourt. "I would not, an I could, have here one single man more than I have," cried Henry.

Our three named doctors, Thomas Morstede, William Bradwardyn, and their medical colleague, Nicol Colnet, must have watched the battle with anxious eyes. I guess that they were stationed during the fight with the baggage animals, and alongside the chaplain who described the action. Their most notable patient would have been the Duke of Gloucester, who was wounded in the abdomen by a dagger thrust. He made a successful recovery.

What happened to the other wounded, one does not know. One suspects that those able to walk made their way back on foot, and that others were taken in country carts captured from the French. The English killed were put into a barn and there burnt. An exception was made with the Duke of York and the Earl of Suffolk, who were among the slain. Their bodies were boiled, so that their bones could be taken home for burial, the Duke of York at Fotheringay and the Earl of Suffolk at Ewelme.

After the death of Henry the Fifth, Morstede became surgeon to Henry the Sixth. He was a Sheriff of the City of London, and a supervisor of surgery, and is said to have written a goodly book on surgery; but if he did, all trace of it is lost. He died in 1450, and was buried in the Church of St. Olave Upwell in the Jewry. His apprentice, Roger Brynard, inherited under his will his surgical instruments and ten marks sterling.

William Bradwardyn was a colleague of Morstede, though probably a little younger, for he was Vice-Master of the enfranchised art of Surgery in London when Morstede was Master, in 1422. So we can imagine that these two surgeons, who were present at the Battle of Agincourt, came back from the war, settled in the City of London, and occupied positions which corresponded in those days to the President and Vice-President of this College.

When Henry the Fifth died, the crown went to his infant son, Henry the Sixth, aged nine months. When he went to France to be crowned in Paris, it was felt necessary to send with him a surgical staff. This is a translation of the recommendation of the Council of the Kingdom:—

May it please your Sovereign Majesty, of your special grace, and on the advice of your wise Council, to ordain that four surgeons take residence in your most honourable household, and to be obedient and answerable to WILLIAM STALWORTH, and to have each of them sixpence a day. And, in addition, to make a grant of twenty pounds sterling for sundry medicines, instruments, dressings (estuffes), and other necessities necessary and pertaining to the office of Surgery for the use of you and the people of your household in this your present journey.³⁴

The names of the four surgeons are not given, but this document is evidence for the presumption made earlier that the surgeon designated as Serjeant Surgeon was in fact the chief surgeon who was responsible for the actions of his colleagues and assistants, and also for the purchase and maintenance of drugs and instruments.

Stalworth evidently did his work well, for he was rewarded with the office of Steward of Cheillesmore, Co. Warwick, with the accustomed fees, wages, and profits.³⁵

The next surgeon of whom we have records is MICHAEL BELWELL, a native of France, who was granted letters of denisation in 1443. No further facts have come to light about this man.³⁶

At the age of 32 Henry the Sixth lapsed into a state of mental imbecility, a legacy perhaps from his French grandfather, and he became incapable of transacting the ordinary business of the realm. In this difficulty the Lords of the Council at Westminster advised the Lord Chancellor to appoint a Commission, consisting of MASTER JOHN ARUNDEL, JOHN FACEBY, and WILLIAM HATCLYFF, physicians, with MASTER ROBERT WAREYN and JOHN

MARSHALL, surgeons, for the purpose of administering and attending to the person of the King. The powers given to these persons were indicated with great care. They were allowed to give the following medicines—electuaries, potions, waters, sirops, confections, laxative medicines, clysters, suppositories, gargles, bathis, head shavings, and many others.³⁷

Whether as the result of the activities of this Commission or not, the King recovered his reason, but only for a spell. In 1455 he relapsed into imbecility, and on this occasion the Dean of Salisbury, MASTER GILBERT KYMER, an "expert proved and notable man in the Craft of Medicine", was sent for to attend the King at Westminster.³⁸ This eminent physician and divine, at one time Chancellor of Oxford University, and author of *Dictarium de Sanitatis Custodia* (D.N.B.), was physician in the household of the King's uncle, Humphrey, Duke of Gloucester, at the time when he was called to attend his sovereign.

Further references to the King's surgeons are wanting, except that in the Regulations for the Household of Henry the Sixth for the year 1454, Robert Waren is cited as serjeant surgeon, John Marshall as yeoman-surgeon, and THOMAS BEKBANK as groom-surgeon.³⁹

In 1461 the imbecile King Henry was deposed, and Edward the Fourth put in his place. One of the acts of the first year of his reign was to make a grant of forty pounds a year for life to MASTER JAMES FRIIS (or FRIIS, or Fryse), his physician. This man was born in the parts of Friesland; he remained in the service of his royal master for twenty years, and received several grants of land and money.⁴⁰

In the second year of his reign the King appointed as his surgeon, MASTER WILLIAM HOBBS, and granted to him some property, including a wharf and crane in the parish of St. Martin in the Vintry, London. In another grant, made eight years later, Hobbs is described as the principal surgeon of the body.⁴¹ Master William Hobbs was evidently a man held in high esteem in the City of London, for in 1461 he was Warden of the Company of Barber Surgeons, and he survived in the troublous times in which he lived to become the body surgeon of Richard the Third.

In 1475, at the head of an army, Edward the Fourth invaded France, but he accomplished little, owing to the astuteness of Louis the Eleventh. There is a picturesque account of the meeting of these two kings on the bridge at Picquigny on the Somme, a town well known to many members of the British Expeditionary Force during the Great War. The medical arrangements for this expedition are preserved in Foedera under the heading of "Wages for the Voyage to France", and so we are able to furnish the actual names and wages of those that served.

Master Jacob Fryse was the physician, receiving as wages two shillings a day. Master William Hobbs, physician and surgeon to the body of the King, had eighteenpence a day, and one surgical assistant at sixpence a day. Then there were seven other surgeons who received one shilling, and five who had only sixpence. The total wages for the medical services for the quarter came to £65 19s. 6d.⁴²

Some at least of these men were of high standing in their profession, for four of them, namely, RICHARD BRIGHTMAN, THOMAS COLLARD, RICHARD

CHAMBERS, and SIMON COLE, at one time held the office of Warden to the Company of Barber Surgeons.⁴³

There are very few records of any sort of the brief reign of the unfortunate King Edward the Fifth, and as far as can be ascertained there is no mention of any medical men.



FIG. 370.—An army surgeon extracting an arrow on the field. Sixteenth century.



FIG. 371.—An army surgeon. Sixteenth century. Note the pointed instrument case near the surgeon's right foot.

Neither is the brief and stormy reign of Richard the Third productive of information concerning members of our profession. There is but one item of information to record, namely, that Richard seems to have continued in office his predecessor's medical attendant, namely, William Hobbys, and granted to him 40 pounds yearly for life.⁴⁴ The name of this surgeon disappears from the records in 1483, and there is naught to show what manner of man he was, nor whether he was near his royal master on fatal Bosworth Field.

It is surprising that the reign of Henry the Seventh is not more productive in records of surgeons—in fact it is very scanty. So far only one surgeon has been discovered,



FIG. 372.—A surgeon's tent near the battle. Note the instrument case hanging in the tent. (Figs. 370-372 are from "Der Arzt und die Heilkunst", by Herman Peters, Leipzig, 1900, by kind permission of Eugen Diederichs Verlag.)

namely, WILLIAM ALTOFTES of Atherston, and of him all we know is that in 1487 he is described as "principal surgeon of the king's body", and that he was given a grant of 40 marks a year for life.⁴⁵ It has been discovered, too, that he was alive in 1520, in the reign of Henry the Eighth, for there is a notice in that year that he was paid his annuity.⁴⁶

As would be expected from a man of his character, Henry the Eighth appointed his own surgeons, and his first was MARCELLUS DE LA MORE, who,

in 1510, was described as the King's surgeon and was granted 40 marks yearly, during pleasure. He seems to have proved satisfactory, for three years later, in 1513, he was made "serjeant of the King's surgeons, with wages and precedence as customary since the reign of Edward the Fourth". More must have been closely associated with the King's physician, DR. THOMAS LINACRE, the founder of the College of Physicians. One likes to think that the relations between the physicians and surgeons were as friendly as they are now, and that more received an invitation to be present at the ceremony of the foundation.

The fact that Marcellus de la More is cited as serjeant of the King's surgeons implies naturally that there were others, and a search in the accounts of the royal household reveals that in 1516 there was a certain JOHN VERARY, who is described as chief surgeon at a

salary of £61 13s. 4d. for life, and in 1509 there is a brief notice of a payment of £61 13s. 4d. to Mr. JOHN, the King's surgeon.⁴⁷

In 1519 Henry seems to have appointed another surgeon in the person of ANTHONY CHABO, a surgeon of London, with an annuity of 20 pounds, and he retained his office at least as long as 1523.⁴⁸

THOMAS VICARY (*Fig. 374*).—The direct successor to Marcellus de la More in the office of serjeant surgeon was Thomas Vicary. The following are the terms in which his appointment is described:—

THO. VICARIE.—To be serjeant of the King's surgeons, and chief surgeon to the King, with allowances both "le bouge the Courte" of the King's household, and of wine, etc., for cures; which office of serjeant was granted by patent 6 Aug. 5 Henry 8. to Marcellus de la More, chief surgeon to the King. Also grant, in reversion, to the said Tho. of the annuity of 40 marks which was granted to the said Marcellus by patent 3 No. 7 Henry 8., to hold from the time when the said office shall be vacant.

It is possible now to trace a direct sequence of these Court Physicians and Surgeons from the time of Edward the Confessor to Thomas Vicary. We

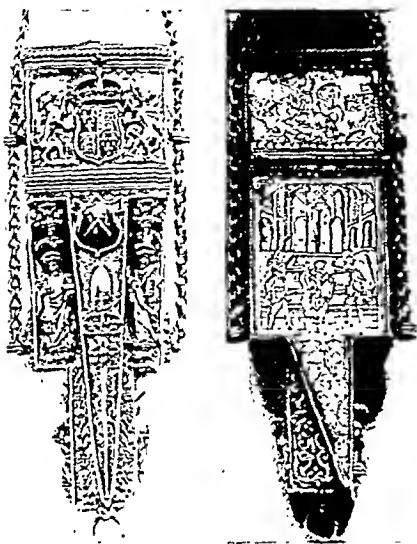


FIG. 373.—A silver instrument case bearing the royal Tudor arms and the arms of the Barbers Company. It may have belonged to Thomas Vicary. (*In the possession of Messrs. Creighton Bros.*)

have seen how in early times they were dignitaries of the Church, how later they became secular and gradually more and more specialized. We have seen the important part they took in the medical arrangements for war; how their duties and privileges, at first vague, were defined. Though they



FIG. 374.—Thomas Vicary.

left no writings behind them, they were the leading men of their time and we should not lose sight of them. In honouring their names, we honour this College, whose aim it is to preserve the best of those traditions which have been handed down to us.

Grateful acknowledgements are made to Sir D'Arey Power, R. R. James, Esq., C. J. S. Thompson, Esq., Messrs. Creighton Bros., and particularly to the Staff of the London Library for their unfailing courtesy and co-operation.

REFERENCES.

- ¹ *Close Rolls*, King John.
- ² *Cal. Patent Rolls*, 1233, 17 Henry III.
- ³ *Cal. Patent Rolls*, 1241, 26 Henry III.
- ⁴ *Cal. Close Rolls*, 1243, 27 Henry III.
- ⁵ *Cal. Close Rolls*, 1248, 32 Henry III.

- ⁶ *Cal. Patent Rolls*, 1253, 37 Henry III.
- ⁷ *Cal. Patent Rolls*, 1251, 35 Henry III.
- ⁸ *Cal. of Documents relating to Ireland*, vol. i, 3106, 1250 to 1251.
- ⁹ *Cal. Close Rolls*, 1251, 35 Henry III.
- ¹⁰ *Cal. Patent Rolls*, 1253, 37 Henry III.
- ¹¹ *Cal. of Charter Rolls*, 1255, 39 Henry III.
- ¹² *Cal. Patent Rolls*, 1256, 40 Henry III.
- ¹³ *Cal. Patent Rolls*, 1260, 44 Henry III.
- ¹⁴ *Cal. Patent Rolls*, 1281, 9 Edward I.
- ¹⁵ *Cal. Patent Rolls*, 1271, 55 Henry III.
- ¹⁶ *Cal. Patent Rolls*, 1279, 7 Edward I.
- ¹⁷ WILLIAM RISHANGER, *Chronica Monasterii S. Albani*, 2nd ed. (H. T. Riley), Rolls Series, 1865, 185-8.
- ¹⁸ NICHOLAS TRIVET, *Annales* (English Historical Society), 371-3.
- ¹⁹ *History of Scottish Medicine to 1860*, 1927.
- ²⁰ *Liber Quotidianus Contrarotulatoris Garderobæ* (Soc. of Antiquaries), 1787.
- ²¹ *Cal. of Doc. relating to Scotland*, 1887, vol. iii, 142.
- ²² T. F. TOUT, *The Place of the Reign of Edward II in English History*, 1914.
- ²³ *Cal. Patent Rolls*, 2 Edward III.
- ²⁴ "The Medical Staff of King Edward the Third", *Proc. Roy. Soc. Med.*, 1925, Feb. 18.
- ²⁵ *William Salt Archaeological Soc.*, vol. xviii.
- ²⁶ G. E. GASK, "The Medical Staff of King Edward the Third", *Proc. Roy. Soc. Med.*, 1925, Feb. 18.
- ²⁷ JOSHUA BARNES, *History of Edward III*, 1688, p. 363. Cambridge.
- ²⁸ FRENCH ROLL, 20 Edward III (William Salt Arch. Soc.), vol. vii, 107.
- ²⁹ *Calendar of Wills in the Court of Husting, London* (edited by R. Sharpe), Part 2, 275.
- ³⁰ *Cal. Patent Rolls*, 5, 6, 7, 10, 13, 15 Richard II.
- ³¹ *Cal. Patent Rolls*, 6 Henry IV.
- ³² *Cal. Patent Rolls*, 11 Henry IV.
- ³³ G. E. GASK, "The Medical Services of Henry the Fifth's Campaign", *Proc. Roy. Soc. Med.*, 1923, vol. xvi.
- ³⁴ *Foedera*, vol. x.
- ³⁵ *Cal. Patent Rolls*, 13 Henry VI.
- ³⁶ *Cal. Patent Rolls*, 21 Henry VI.
- ³⁷ *Proceedings of the Privy Council*, vol. vi.
- ³⁸ *Foedera*, vol. xxi.
- ³⁹ *Proceedings of the Privy Council*, vol. vi.
- ⁴⁰ *Cal. Patent Rolls*, 1, 3, 13, 21 Edward IV.
- ⁴¹ *Cal. Patent Rolls*, 2, 10 Edward IV.
- ⁴² *Foedera*, vol. xi.
- ⁴³ *Annals of the Barber Surgeons Company*.
- ⁴⁴ *Cal. Patent Rolls*, 1 Richard III.
- ⁴⁵ *Cal. Patent Rolls*, 2 Henry VII.
- ⁴⁶ *Letters and Papers, Foreign and Domestic*, Henry VIII.
- ⁴⁷ *Ibid.*, 1, 2, 5, 6, 7 Henry VIII.
- ⁴⁸ *Ibid.*, Henry VIII, vol. iii.
- ⁴⁹ *Ibid.*, vol. iv, part 3, 2857, 21 Henry VIII.

THE TREATMENT OF GLIOMATA AND PITUITARY TUMOURS WITH RADIUM.

By SIR PERCY SARGENT,

SENIOR SURGEON TO ST. THOMAS'S HOSPITAL, LONDON :

AND STANFORD CADE,

ASSISTANT SURGEON TO WESTMINSTER HOSPITAL, LONDON.

Of all malignant tumours the gliomata would, *a priori*, seem to be the most favourable for radium treatment. They do not infiltrate tissues other than those of the central nervous system itself, and remain, almost without exception, within the confines of the meninges: they give rise to no metastases; they do not invade lymphatic glands. Further, they are, for the most part, composed of cells of a primitive character.

It is, perhaps, convenient for the purposes of this paper to adopt some of the terms employed by Bailey and Cushing¹ in their attempt to classify the gliomata, as some of them have already, though perhaps prematurely, passed almost into general use. We recognize, however, that that classification, and indeed some of the terms, must at present be only provisional, just as are certain other names given to the same tumours by previous workers, notably Greenfield.² The types of glioma which we have so far had the opportunity of treating with radium are, according to the nomenclature of Bailey and Cushing, spongioblastoma, oligodendroglioma, medulloblastoma, and astrocytoma. It is to Dr. Greenfield that we are indebted for all histological examinations and opinions, without which this paper would have little value.

Before we undertook the observations upon which the paper is based, one of us had employed radium in a number of cases of glioma, quite empirically, and with little to guide either the mode of application or the dosage. Nevertheless the results were such as to justify the general conclusion that under the influence of radium gliomata were apt to behave in an unusual manner, in some cases unexpectedly to recede, remain inactive, or even apparently to disappear. One example will suffice to support this generalization.

A professional man, 47 years of age, came under the care of Dr. Gordon Holmes with a history of cerebellar symptoms extending over three months. In September, 1921, the cerebellum was exposed, and a mass of soft, gelatinous tumour was removed from within the right hemisphere. He made a good recovery, but symptoms recurred three months later, and a second operation was performed. Masses of growth were removed, and 62 mgrm. of radium, screened with 2 mm. of lead, introduced into the middle of the growth. The radium was left in position forty-eight hours. The total dose given was 2976 mgrm.-hours (Dr. Martin Berry). After a stormy convalescence the patient made an excellent recovery, and remains well and almost free from symptoms after nine years.

HISTOLOGY.—This is a very cellular tumour, with cells of two distinct types. Some are large with abundant cytoplasm and indefinite processes passing some

distance from the cell body. In some cases the processes of one cell seem to fuse with those of another, but this is not very clear. The nucleus of these cells is large, vesicular, and palely staining, with a central or paracentral nucleolus. The other cells are small, with round, dark-staining nuclei and very little cytoplasm. The blood-vessels are numerous, with fairly thick walls, usually showing endothelial hyperplasia. Mitoses are numerous in some parts. The tumour tissue removed at the two operations has identical characters. The large cells with clear nuclei may be neuroblasts. If so, the tumour would correspond with Bailey and Cushing's 'medulloblastoma neuromatosum'.

We are aware, of course, that quite apart from radiation treatment some patients with gliomata survive for many years, and that some types of glioma have at least a very low degree of malignancy. But the case quoted is one, not of survival, but of apparent cure with partial restoration of cerebellar function.

PRINCIPLES OF IRRADIATION.

The treatment of cerebral tumours by radium is based upon the same principles as guide the clinician in all radium therapy. The fundamental element is the variation in the response between normal neuroglial tissues and that of tumours. Both from the study of clinical cases and from experimental irradiation of animals' brains, it is evident that nerve tissue is resistant to the effect of gamma rays to such a degree that within therapeutic dosage no caustic effect is obtained. The effect on the tumour cells is inhibitory to growth, and the combination of sensitivity of the neoplasm and resistance of normal brain offers a field of therapy well worth exploring. It is not denied that destruction of tissue in the immediate vicinity of the source of irradiation can be produced experimentally³; necrosis so obtained is due to inadequate filtration and the effects of beta particles, or to an excessive intensity of gamma irradiation.

In our own experiments necrosis has been obtained by using radon seeds of 3- or 5-mc. strength screened by 0.3 mm. of platinum or silver or by insertion of a 10-mgrm. tube left in position for ten days. The experimental evidence was obtained by the introduction of radon seeds and radium needles into the cerebral hemispheres of rabbits. The effect of variations in screenage, dosage, and time were studied. The rabbits were killed at varying intervals—from a fortnight to five months—after removal of the radium. In some cases the radon seeds were left *in situ* permanently. These experiments showed that necrosis occurred in the immediate vicinity of the source of irradiation and it did not extend beyond a few millimetres from it; necrosis was directly proportional to the dosage and inversely proportional to the screenage. It is obvious that some of the changes were due to the trauma of the needling, as we know that they can be produced by the use of dummy needles or empty seeds. The effect of trauma can be entirely eliminated by placing the radium between dura and bone, or better still by employing surface irradiation. After recovery from operation the animals appeared to be normal in every respect. A full account of these experiments will be published later. The experiments go to prove that irradiation of the brain requires as much care, thought, and attention to details as irradiation elsewhere. The success of treatment depends primarily upon the histological nature of the

tumour, and secondly upon accurate and correct irradiation. The first condition is at present beyond clinical control, although methods of increasing the radiosensitivity of tumours are being tried, and not without some encouragement. The second condition depends upon technique of irradiation. Reduced to its principles, it depends upon the linear intensity, the screenage, and the time factor. A low linear intensity, pure gamma irradiation, and a prolonged time factor, give the patient a greater chance of recovery.

CHOICE OF METHOD OF IRRADIATION.

The two methods at the disposal of the surgeon are interstitial and surface irradiation. The choice of the method depends upon two factors: (1) The localization of the tumour; (2) The operability. Tumours situated in the pituitary region are not suitable for surface radium therapy. Unlocalized tumours, or those situated near the base of the skull, the mid-brain or near the medulla, are not suitable for interstitial irradiation. The ideal, however, is to submit each case to a two-stage treatment combining interstitial irradiation with surface treatment whenever possible, and employing high-voltage X-ray therapy as a second stage in cases when surface therapy is not practicable.

Decompression.—Preliminary decompression is an essential step of the treatment. It offers relief from increased intracranial tension, whilst a window is created through which the underlying tumour can be irradiated without the screenage offered by bone, an appreciable factor in some skulls; the risk of radium osteo-necrosis is eliminated; removal of the tumour becomes possible in some cases, and even partial removal of a tumour is of definite benefit; a biopsy is obtained; and interstitial irradiation can be carried out.

INTERSTITIAL IRRADIATION.

Needles.—The use of needles containing small quantities of radium (1 to 2 mgrm. per needle) is preferred to the use of radon seeds in all situations with the exception of the pituitary. Needles of 27 or 33 mm. over-all length are employed; accurate distribution of the needles within and around the neoplasm is possible (*see Figs. 383–385*); to obtain such distribution with seeds is a feat rarely accomplished. The falling intensity of emanation is a factor of equal importance, and in the treatment of gliomata it is a factor which cannot be overestimated. The needles, threaded with carbolized silk, are inserted equidistally 1 cm. apart, great care being taken to avoid blood-vessels. All the threads are tied together and cut short; the knot is placed between the dura and the flap, only one thread being brought out through the incision. The wound is closed without drainage. The needles are left in position for seven days, the wound is then partially re-opened, and with the single thread as a guide the knot is found and the needles are removed. An alternative method used occasionally is to employ silver wire instead of thread and to bring all the wires outside the incision (*see Fig. 386*).

Seeds.—The use of seeds is indicated in the pituitary region (*see Figs. 389–392*). Very great caution must be exercised in the irradiation of this area; two or three seeds each containing 1.2 mc. screened by 0.5 mm. of

platinum are inserted. The seeds tend to fall to the bottom of the sella, and unless a great many seeds are used, irradiation is not evenly distributed. The possibility of producing permanent blindness by excessive dosage must be kept in mind. In connection with the dosage of radon which may be used in pituitary tumours, we have been given the opportunity of making use of the following case :—

M. S., female, age 25, was first seen at St. Thomas's Hospital in July, 1928, with bitemporal hemianopia and headaches of eighteen months' duration. She was operated upon in October, 1928, when, through a transfrontal osteoplastic approach, a quantity of growth was removed from a pituitary tumour, which proved



FIG. 375.—Case M.S. Illustrating ten seeds in the region of the optic chiasma.

to be a so-called adenoma (adenomatous hyperplasia). At the same time ten radon seeds were inserted into the cavity ; these were subsequently found radiographically to be aggregated into a single mass close to the optic chiasma (*Fig. 375*). The visual fields improved to a slight, but quite definite, extent for five months, but ten months after the operation they were found to have deteriorated again. The discs were found to be atrophic, and there were no hæmorrhages. Accordingly, in September, 1929, a second operation was performed. The original flap was again turned down, and the left frontal lobe elevated so as to expose the sellar region. There was no increased intracranial pressure. The seeds, together with a "mass of white tissue resembling slough", were removed. This tissue showed "excessive glial proliferation ; thrombosis ; no neoplastic tissue". The patient made a good recovery in every way except as regards vision, which steadily deteriorated until complete blindness resulted (*Figs. 376-379*).

This case is of the greatest interest from the point of view of the number of seeds to be employed, and the difficulty of spacing them in the soft, or even cystic, masses which constitute the majority of pituitary tumours. We ourselves have never employed more than four seeds, each with 1.5 mc. screened by 0.5 mm. of platinum or gold. Here ten seeds were used, each of

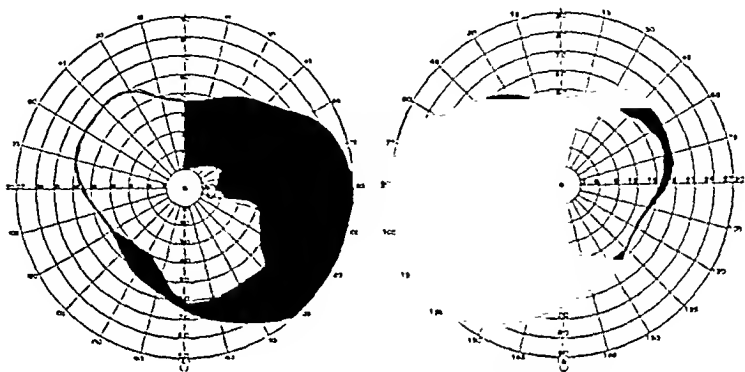


FIG. 376.—Case M.S. Visual fields before operation.

2 mc. screened by 0.3 mm. of platinum, and they were aggregated close to the optic chiasma. In spite of this the vision improved for some months before commencing the deterioration which ended in blindness (*see Figs. 376-379*). We are not prepared to say whether this was a late result of placing what we consider to be a non-therapeutic dose of radon close to the optic

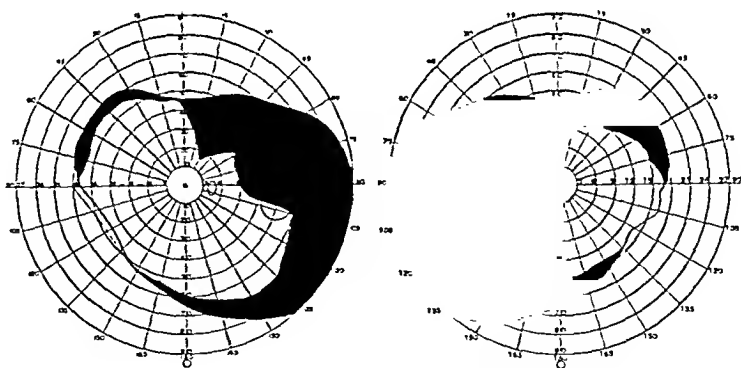


FIG. 377.—Case M.S. One month after insertion of radium.

chiasma, or an indirect effect upon the visual paths by causing the glial proliferation noted later in their neighbourhood. Possibly neither of these suggestions embodies the true explanation, which might equally well be that the left eye had already been so badly damaged before the first operation that eventual recovery of vision was impossible; whilst the adenomatous mass may still have been encroaching upon the right side of the chiasma, the

operation having been done upon the left side. It is our experience that the vision improves most upon the side operated upon, and it is our practice, therefore, always to operate upon the side of the better eye.

It is evident from the case reported above that some effort had to be made to ensure an even distribution of irradiation over a wider area and giving more

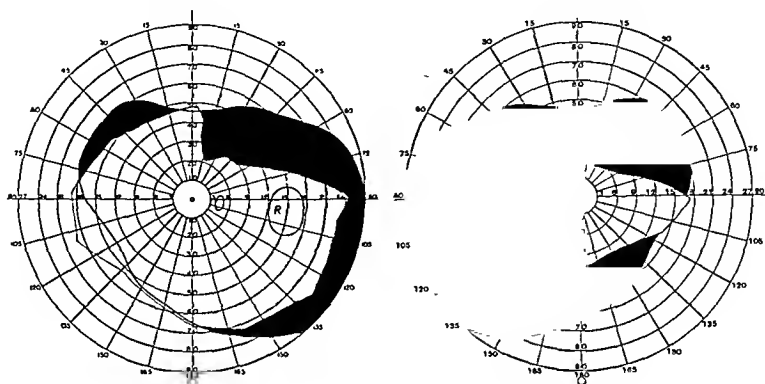


FIG. 378.—Case M.S. Five months after insertion of radium.

control as to the position of the radio-active focus. To secure this object a special container was made for us by Dr. Alton, of the London Radium Institute. Its object is to provide a source of irradiation filling the sella tureica and projecting upwards into the suprasellar space. It is made of silver (1 mm.), is oblong, and is filled with the desired amount of radon; 8, 10,

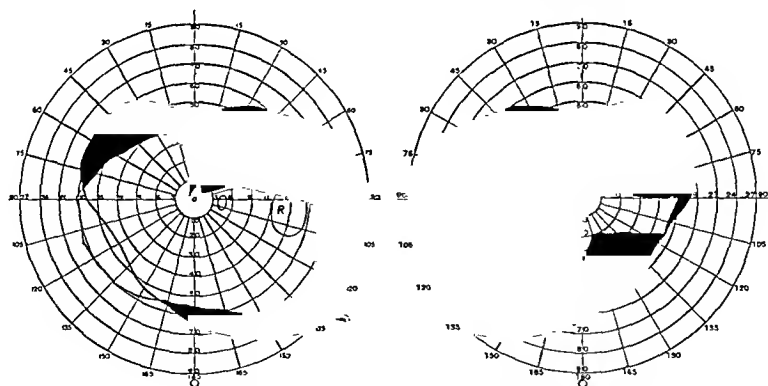


FIG. 379.—Case M.S. Eight months after insertion of radium.

or even 15 mc. can be used. The peripheral area irradiated is four to six times as wide as that irradiated by two or three separate seeds (*see Figs. 393, 394*).

For interstitial irradiation of tumours of the cerebellum seeds present certain mechanical advantages over needles (*see Fig. 387*).

Immediate Effects of Interstitial Irradiation.—Healing of the wound is slower than normal; leakage of cerebrospinal fluid may continue for several

weeks. If radium is placed above the tentorium, there are no untoward symptoms of any importance, though papilloedema is as a rule increased and retinal hæmorrhages may occur, though not so commonly as with surface treatment. Below the tentorium, interstitial irradiation is not so well tolerated; headache is very common, persistent hiccup has been noted in several cases, polyuria is the rule, and escape of cerebrospinal fluid may be continuous.

SURFACE IRRADIATION.

The method employed is that of a skull-cap of 'sorbo' rubber, or 'columbia paste' (Figs. 380, 381). It is applied as soon as the operation wound is soundly healed. The radium-skin distance is 15 mm. The quantity of radium varies from 50 to 75 mgrm. in numerous needles. The area treated is wide, half the head (right or left side), or the occipital or frontal areas, according to localization. With this quantity of radium it is possible to obtain an inhibitory effect on tumours situated near the corpus callosum, in the lateral ventricles, or encroaching on the mesial aspect of the cerebral hemisphere. That a cap so constructed, carrying 50 mgrm. of radium, emits irradiation reaching a depth of 4 or 6 cm. has been proved experimentally by Dr. W. G. Evans, who, working in conjunction with one of us, carried out the following experiment: An accurately fitting cap was made and applied to a cadaver after sagittal bisection of the head and insertion of an X-ray film between the two hemispheres. At the end of four hours the film when developed showed obvious evidence of having been submitted to irradiation. These experiments will be published in full elsewhere.

By this method a large area of the brain can be treated. The treatment is intermittent, fourteen to eighteen hours being given daily for a period of from two to three weeks in cases where a previous interstitial treatment has been given, and from four to six weeks if surface application is the sole method employed.

Immediate Effects of Surface Irradiation.—At first the papilloedema is increased, but after seven to ten days this diminishes. Retinal hæmorrhages have occurred in so many cases that they are now looked for and expected; but their significance is not yet understood.

The effect on the secretion of cerebrospinal fluid is interesting. It is known that X-radiation to the head decreases the activity of the choroid plexus; there is evidence that a similar effect is obtained by radium, but

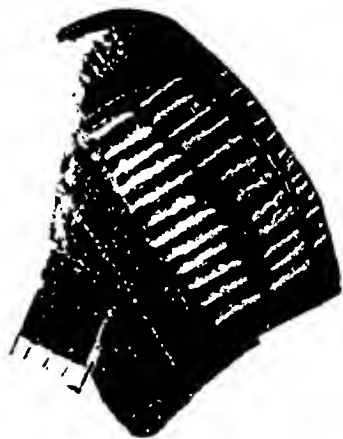


FIG. 380.—'Sorbo' rubber cap for treatment of a tumour in the right fronto-parietal region, and showing the distribution of the needles.

further study is necessary to obtain definite evidence. After operations upon the brain there is an increase in the production of cerebrospinal fluid. This may be due to hypersecretion resulting from the manipulation, and so be similar to the increase which can be produced experimentally by the administration of brain extract. The increase at times is so great that repeated



FIG. 381.—A, 'Sorbo' rubber cap for surface treatment of tumour in the temporal region. B, Skiagram showing the distribution of the needles in relation to the tumour and the skull. C, Skiagram showing portion of bone removed at operation. D, The same, illustrating the area under the influence of the radium; the clear area is produced by the radium and corresponds to the position of the bony opening as shown in C.

tapping or lumbar puncture is necessary. In the majority of cases there is a gradual diminution of the size of the swelling, and in all successful cases the area of the flap becomes concave. Surface irradiation is continued until all the hair is shed, and in some cases until peeling of the scalp results. In the latter event the regeneration of hair follicles does not take place.

Dosage.—The criticisms of the 'milligram-hour' method of notation are numerous and in some ways justifiable, but the alternative methods 'erg-cubic-centimeter milligram-hour' of John Murdoch or 'skin erythema dose' of Quick, Failla, and Quimby are not universally accepted. All methods of notation are faulty, but until such a time as a universal unit of measurement is available, we prefer to note the dose in 'milligram-hours', giving the necessary data of number of foci, amount of radium, time, screenage, distance, and method of irradiation.

CASE REPORTS.

The following twenty-one cases were selected to illustrate the type of tumour treated and the methods employed. They do not represent any particular histological type, but rather a mixed group illustrating failures and successes, pointing out the dangers of the treatment, the disappointing end-results in some, and apparent successes in others. A series of cases treated by surface irradiation by one of us is not included, although the results are most encouraging, as the study of that series is not yet completed. The few cases treated by teletherapy (the so-called radium 'bomb') are also omitted, as it is not desirable at present to express any opinion on this method of radium therapy.

TUMOURS OF THE CEREBRUM.

Case 1.—A. H., male, age 37 years. (Dr. J. P. Martin.)



FIG. 382.—*Case 1.* Oligodendroglioma showing calcification in the frontal and parietal area.

HISTORY.—Two years. Frontal headache; vomiting; ten attacks of loss of consciousness; blurring of nasal edges of both discs.

X RAY (Fig. 382).—Definite irregular opacity in left fronto-parietal region.

OPERATION (Sept. 27, 1929).—Left parieto-temporal opening, and large sub-cortical tumour exposed. The tumour was found to be deeply infiltrating the brain; the main mass of the tumour, which was the size of a small tangerine orange, was removed, but the deepest part was not reached. The bone-flap was removed and the skin closed.

HISTOLOGY.—Oligodendroglioma.

RADIUM TREATMENT (Nov. 8).—Eighteen needles each containing 1.3 mgrm. of radium element, screened by 0.6 mm. of platinum, were inserted into the periphery



FIG. 383.—Case 1. Showing decompression and eighteen radium needles in position.

of the tumour (Fig. 383) and left *in situ* for 7 days. Total, 3721 mgrm.-hrs. This was followed by surface application of 80 mgrm. of radium at a distance of 15 mm. by means of a columbia-paste cap, 16 hours daily for 10 days. Total, 12,960 mgrm.-hrs. There was marked erythema and the hair rapidly fell out.

AFTER-HISTORY.—The patient returned to work and remained well and active for one year; he died suddenly in November, 1930, but no details could be obtained.

Case 2.—K. H., female, age 49 years. (Dr. Gordon Holmes.)

HISTORY.—One and a half years. Attacks of momentary faintness. Recently loss of consciousness. Three weeks' pain in the eyes and forehead; vomiting. Persistent frontal headaches. Bilateral papilloedema; left internal strabismus.

OPERATION (Nov. 8, 1929).—Left temporo-parietal decompression. Subcortical tumour removed from the posterior end of the second temporal gyrus. The tumour was red, soft, and the size of a hen's egg.

RADIUM TREATMENT.—Seven days after operation 7 needles each containing 2 mgrm. of radium, and 3 needles each containing 1.3 mgrm., were inserted into and around the site of the tumour interstitially. The radium was left in position for 12 days. Total, 5184 mgrm.-hrs. On Jan. 10, 1930, a skull-cap made of columbia paste carrying 35 mgrm. of radium was applied for 12 hours daily for 20 days. Total, 10,900 mgrm.-hrs.

HISTOLOGY.—Glioma of rather unusual structure owing to the number and

large size of rosette arrangements, in which elongated cells are grouped with their nuclei arranged almost in palisade formation, and a long process extending inwards into a non-nucleated area. Cells chiefly bipolar. Mitotic figures common.

AFTER-HISTORY.—Three months later the patient was clinically improved, but the hernia cerebri was very tense. She died from the disease five months after treatment.

Case 3.—J. B., female, age 13 years. (Dr. Kinnier Wilson.)

HISTORY.—Ten months. Astereognosis in left limbs: papilloedema; left homonymous hemianopia.

X RAY.—Showed a tumour, calcified in parts, in the right occipital area.

OPERATION (Dec. 7, 1929).—Right-sided osteoplastic flap. Tumour removed from occipital lobe.

RADIUM TREATMENT.—Thirteen needles were inserted—9 needles each containing 1.3 mgrm. of radium, screened by 0.6 mm. of platinum, into adjacent brain



FIG. 384.—*Case 3.* Antero-posterior view showing thirteen needles in position. Of these, nine are around the periphery and four in the centre of the space previously occupied by the tumour.

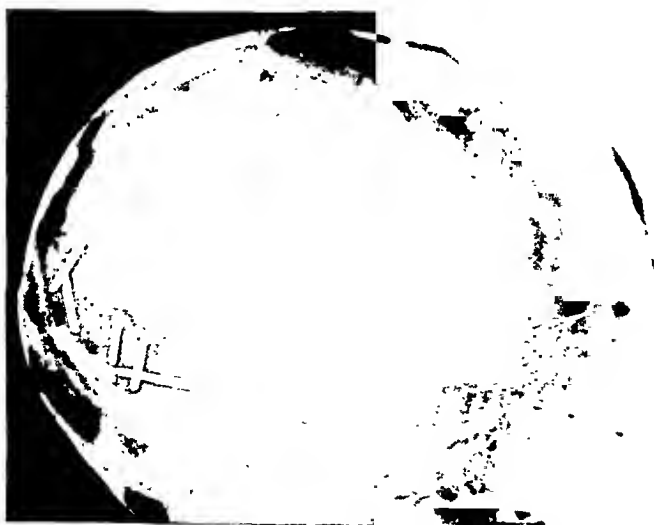


FIG. 385.—*Case 3.* Lateral view, showing radium needles in position.

posteriorly; 4 needles each containing 2 mgrm., screened by 0.65 mm. of platinum, into the space previously occupied by the tumour (*Figs.* 384, 385). The needles were left in position for $4\frac{1}{2}$ days. Total amount of radium used, 20 mgrm.; period of irradiation 96 hours. Total interstitially, 1917 mgrm.-hrs. Surface irradiation by means of a columbia-paste cap on Jan. 14, 1930. Total quantity of radium used, 70 mgrm., screened by $\frac{1}{2}$ mm. of platinum and $\frac{1}{2}$ mm. of brass. Daily application 8 hours for 12 days, with occasional interruptions in the treatment. Total, 5180 mgrm.-hrs. The hair came out and there was definite erythema.

HISTOLOGY.—Oligodendroglioma.

AFTER-HISTORY.—The patient remains well to date.

Case 4.—E. C., male, age 43 years. (Dr. Hinds Howell.)

HISTORY.—Severe headaches, especially at the back of the head. Unsteady gait. Papilloedema about four dioptries. Nystagmus. Motor power good, and equal on both sides.

X RAY.—Showed large irregularly-shaped tumour in the left parietal region.

OPERATION (Feb. 28, 1930).—Left parietal flap. Discoloured tumour in the brain substance lying in the parietal lobe behind the post-central gyrus. Mass of tumour as big as a hen's egg removed. The tumour had a pedicle which passed far into the substance of the brain in the direction of the corpus callosum.

HISTOLOGY.—Oligodendroglioma.

RADIUM TREATMENT.—Six days after operation 10 needles each containing 1.33 mgrm. of radium, 27 mm. over-all length, screened by 0.6 mm. of platinum, were inserted into the periphery of the tumour and left in position for 7 days. Total, 2226 mgrm.-hrs.

AFTER-HISTORY.—The patient remained well for ten months. He was then re-admitted to hospital with a tense hernia cerebri and recrudescence of symptoms. He is at present under treatment.

Case 5.—E. K., female, age 39 years. (Dr. Gordon Holmes.)

HISTORY.—The history is difficult to get and is incomplete. Five months' vomiting and three months' headache. Inability to fix her attention. Drowsy. No fits of any kind. Diminution of sensation on the left side.

X RAY.—Skiagram of head negative.

OPERATION (Jan. 31, 1930).—Large right parietal flap. Dark-red infiltrating tumour on the lower lateral surface of the frontal lobe reaching the surface above and in front of the Sylvian point, infiltrating the brain extensively. Much tumour tissue was removed with very little loss of blood.

RADIUM TREATMENT.—Twelve mgrm. of radium were inserted at the periphery and 6 mgrm. into the cavity previously occupied by the tumour. Total, 19.3 mgrm.

AFTER-HISTORY.—The patient died in coma forty-eight hours after operation. Death from cardiac failure.

POST-MORTEM.—The anterior end of the corpus callosum appeared to be infiltrated with tumour, and farther back the infiltration reached the caudate nucleus. Histologically the tumour is a glioma which varies very much in structure, many cells being collected in clusters or small rosettes. In other places the cells are loosely scattered in a matrix of neuroglial fibres.

Case 6.—J. P., male, age 13 years. (Dr. Gordon Holmes.)

HISTORY.—Four months. Headaches, vomiting, increased irritability; no loss of power or wasting. Reflexes normal. Weakness of right external rectus. Slight weakness of left hand.

X RAY.—No abnormality detected.

OPERATION (April 8, 1930).—Right temporal decompression. Removal of a large tumour from the right temporal lobe.

RADIUM TREATMENT.—Fourteen needles each containing 1.3 mgrm. of radium were inserted at the periphery of the cavity previously occupied by the tumour, and 10 mgrm. into the cavity.

AFTER-HISTORY.—The patient died thirty-six hours after operation.

HISTOLOGY.—Glioblastoma.

Case 7.—M. S., female, age 24 years. (Dr. Adie.)

HISTORY.—Four months. Headaches, vomiting, dimness of vision; amenorrhœa for three months. Bilateral papillœdema of about three dioptries in each eye, with some hemorrhages. Left pupil larger than right. Muscular power and tone good. Diminished sensation to pin-prick on left side of body. The patient has had four attacks of unconsciousness associated with convulsive movements of jaw and clonic contractions of arms.

X RAY.—No evidence of any abnormality.

OPERATION (Feb. 10, 1930).—Right temporal decompression (Mr. Donald Armour). Subcortical tumour the size of a large oyster removed from the temporal area. A month after operation there was no diminution of papillœdema.

HISTOLOGY.—Astrocytoma fibrillare.

RADIUM TREATMENT.—A columbia-paste cap carrying 55 mgrm. of radium in 34 needles was applied at a distance of 15 mm. from the skin. The patient received 18 hours daily, and treatment continued for 21 days. Total, 20,590 mgrm.-hrs. Total loss of hair on the side treated and over a small area on the opposite side. The skin peeled on the side treated, papillœdema disappeared, and the general condition of the patient improved. There were no fits.

AFTER-HISTORY.—The hernia cerebri has entirely disappeared; the operation area is concave; there is no papillœdema; menstruation has returned. She has had three fits since leaving hospital.

Case 8.—G. A., female, 34 years. (Sir James Purves-Stewart.)

HISTORY.—Thirteen months. Headaches and vomiting; momentary blindness; deterioration of vision; papillœdema; dragging of right leg. Fits.

X RAY.—Pneumoradiogram showed both lateral ventricles displaced to the right.

OPERATION.—Left frontal decompression (Mr. Arthur Evans). Tumour not removed. Following operation: aphasia, right-sided weakness, incontinence, and marked hernia cerebri.

RADIUM TREATMENT.—Columbia-paste plaque applied: 50 mgrm. of radium. Continuous application for 24 hours followed by 12 hours daily for 10 days. Total, 6600 mgrm.-hrs. Peeling of skin over forehead and loss of hair.

AFTER-HISTORY.—Complete recovery with the exception that vision in the left eye is reduced to perception of light. Well and symptomless three and a half years later.

Case 9.—W. W., male, age 61 years. (Sir James Purves-Stewart.)

HISTORY.—Right homonymous hemianopia; hemi-anæsthesia; right ankle-clonus; Jacksonian fits involving right upper and lower limbs.

OPERATION (March 12, 1926).—Left parieto-occipital osteoplastic flap (Mr. Arthur Evans). Subcortical tumour present in left occipital lobe.

HISTOLOGY.—Gliosarcoma.

RADIUM TREATMENT (March 25, 1926).—Columbia-paste cap 21.85 mgrm. applied 16 hours daily for 16 days. Total, 5632 mgrm.-hrs. The patient recovered and remained well till November, 1926, when he had several fits. A second radium treatment was given, 50 mgrm., 14 hours daily for 15 days, then the amount was reduced to 20 mgrm. for a further 6 days. Total, 14,700 mgrm.-hrs.

AFTER-HISTORY.—The patient remained well for twelve months, then died after a fit.

Case 10.—A. W., male, age 9. (Dr. Gordon Holmes.)

HISTORY.—Five years' history of fits recently getting more frequent and worse: for three years associated with loss of consciousness.

OPERATION (May 10, 1930).—Frontal decompression. Much solid growth removed. Uneventful recovery.

HISTOLOGY.—Fibrillary glioma.

RADIUM TREATMENT.—Seven days after operation 12 needles of 1 mgrm. were inserted into the brain at the periphery of the tumour and into the centre (*Fig. 386*). Radium left in position 7 days. Total, 2016 mgrm.-hrs.



FIG. 386.—*Case 10*. Showing radium needles placed in and around tumour-bed; silver wires are attached to the needles.

The cerebellum was incised and a small portion of the tumour removed.

RADIUM TREATMENT.—Eight gold seeds at 1.5 mc. each, screened by 0.5 mm. of gold, inserted (*Fig. 387*). The patient died seven hours after operation.

POST-MORTEM.—Examination revealed that the fourth ventricle was filled with gelatinous growth which extended upwards, dilating the iter of Sylvius and reaching downward to the foramen of Magendie.

HISTOLOGY.—The tumour is extremely reticular in structure in almost all parts, but towards the intact cerebellum, which is infiltrated rather than pushed aside by the tumour, it is more fibrous and frankly gliomatous in structure.

Case 12.—H. P., male, age 32. (Dr. Gordon Holmes.)

HISTORY.—The patient was admitted to hospital twenty months previously with nausea, headaches, staggering gait, diplopia, papilloedema, and coarse lateral nystagmus; hypotonia of both arms; ataxy and dysidiadochokinesis on the left.

FIRST OPERATION.—Partial removal of left cerebellar tumour (Mr. Donald Armour). The patient was re-admitted with a bulging occipital tumour; increasing nausea and staggering and pain in fronto-temporal region.

HISTOLOGY.—Astrocytoma.

SECOND OPERATION (June 10, 1930).—The patient was again operated on by

TUMOURS OF THE CEREBELLUM.

Case 11.—C. R., male, age 12 years. (Dr. Gordon Holmes.)

HISTORY.—Admitted in May, 1930, with headaches, vomiting, diplopia, papilloedema, and definite signs of a right-sided cerebellar tumour.

OPERATION.—Cerebellar decompression. Right cerebellar lobe found to be bulging.



FIG. 387.—*Case 11*. Showing eight gold seeds in cerebellar tumour.

Mr. Armour. The tumour was found filling the bone opening, part of it was cystic, and on the superficial wall of the cyst a number of rounded tumour masses were seen, varying in size from that of a pea to that of a walnut. The tumour was partly removed, two very large pieces being taken away.

RADIUM TREATMENT.—Ten needles of 1 mgrm. each were inserted peripherally around the site of the tumour, and 10 mgrm. into the centre, the threads being brought out of the wound. The radium was left in position for 7 days. Total, 3360 mgrm.-hrs.

HISTOLOGY.—Nature of tumour removed at the second operation: sarcoma rather than glioma.

AFTER-HISTORY (July 5, 1930).—Flaccid paraplegia. Absence of knee- and ankle-jerks. Difficulty in micturition. Two days later the paraplegia was complete, the abdominal reflexes were lost, and there was complete retention of urine. Evidence of tumour formation in the cord with steady deterioration of general condition.

Case 13.—A. S., male, age 14 years. (Dr. Gordon Holmes.)

HISTORY (March 31, 1930).—Three months' history of headaches and vomiting. Previously operated on for cerebral tumour in December, 1928; then history of headache, nausea, vomiting, and failure of vision for twelve months. Bilateral sub-tentorial decompression—cystic tumour opened in right cerebellar hemisphere. On discharge from hospital the neurological examination was eventually negative except for a questionable weakness of the left internal rectus. The patient remained well till three months previous to admission. He then began to vomit, at times incessantly; headaches and swelling on the back of the head (operation area). Discs pale. No retinal hæmorrhages. Motor system, no weakness. Co-ordination normal. Tends to fall to the right.

OPERATION (May 9, 1930).—The old incision was opened and a cystic collection seen. On the medial wall of the cyst was a soft mass 4 by 5 cm., clearly tumour, in the substance of the destroyed right cerebellar lobe. The mass was removed.

RADIUM TREATMENT.—Four needles containing 5 mgrm. of radium element each were laid on the surface of the visible part of the tumour: the threads were led out of the wound (Fig. 388). Total dose in 7 days, 3360 mgrm.-hrs.

HISTOLOGY.—Fibrocellular glioma.

AFTER-HISTORY (November, 1930).—The patient is symptomless, vision is good, there is no motor weakness, and he has returned to school.

Case 14.—F. R., male, age 4½ years. (Dr. Birley.)

HISTORY.—Two months' history of severe headache and vomiting. Bilateral papilloedema.

OPERATION (July 2, 1930).—Cerebellar decompression, glioma in mid-line. Partial removal. Recovery and improvement.

HISTOLOGY.—Glioma with fairly well differentiated neuroglia fibres and large oval nucleus of the type of adult neuroglia cells. The cells are fairly closely placed. The neuroglial cells appear to be fully developed, and the tumour may therefore be classed as an astrocytoma fibrillare.



FIG. 388.—Case 13. Showing four needles in the cerebellar region.

RADIUM TREATMENT.—Two months after operation. Columbia-paste cap made 15 mm. thick, 30 mgrm. of radium in 30 needles applied, 12 hours daily for 14 days. Total, 3600 mgrm.-hrs. Complete recovery of vision and motor function.

AFTER-HISTORY.—Uneventful recovery; remains well, and almost free from symptoms.

Case 15.—L. C., male, age 12 years. (Dr. Birley.)

HISTORY.—Two years' history of mental change, staggering gait, frontal headaches, deterioration of vision, occasional vomiting. Optic atrophy on right side; papilloedema left side. Nystagmus. Inco-ordination of both arms and legs.

OPERATION (June 26, 1924).—Tumour found in deeper portion of right frontal lobe. Partial removal.

RADIUM TREATMENT.—Insertion of 100 mgrm. of radium, left in position for 24 hours. Total, 2400 mgrm.-hrs.

AFTER-HISTORY.—Temporary improvement. Death from recurrence took place seven months later.

PITUITARY TUMOURS.

Case 16.—A. H., male, age 59 years. (Dr. Grainger Stewart.)

HISTORY.—Twelve months' history of failing vision and headache. Bitemporal hemianopia. Visual acuity: right, 6/12; left, cannot count fingers. Discs atrophic.

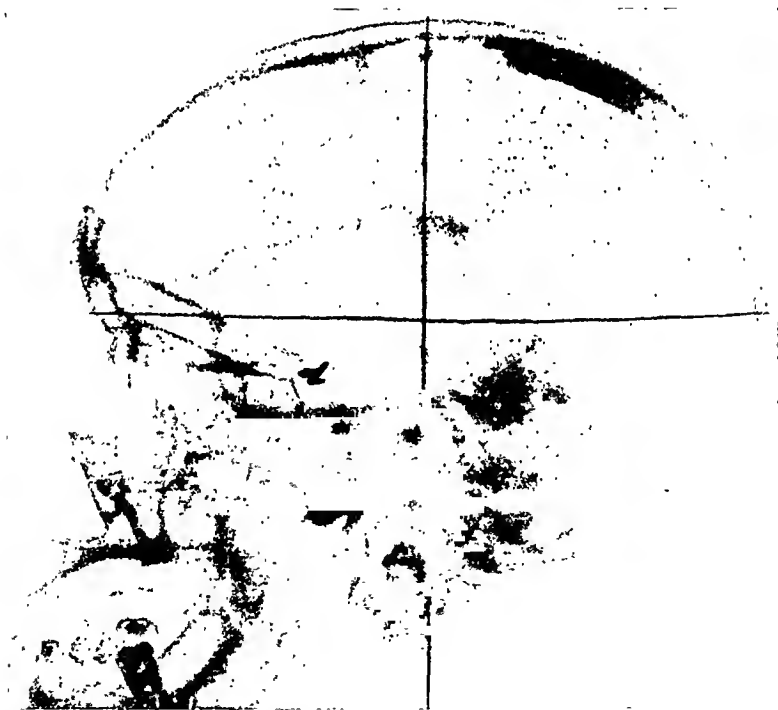


FIG. 389.—*Case 16.* Showing four gold seeds in the upper part of the pituitary fossa. The seeds were introduced through a frontal approach.

X RAY.—Nothing abnormal.

OPERATION (March 22, 1930).—Right-sided osteoplastic frontal flap. A suprapituitary tumour, partly cystic, was exposed, and partially removed.

RADIUM TREATMENT.—Four radon seeds, 1.2 mc. each, screened by 0.5 mm. of gold, were inserted. Total, 633 mgrm.-hrs. (*Figs. 389, 390*).

HISTOLOGY.—Adamantinoma.

AFTER - HISTORY.—The patient remains well to date, but did not come up for examination.

FIG. 390.—*Case 16.*
Antero-posterior view.

Case 17.—Dr. J. W., male, age 35 years. (Dr. Gordon Holmes.)

HISTORY.—Five months' history of failure of vision. Noticed that in reading the middle part of the word would be left out. Bitemporal hemianopia, and X-ray evidence of erosion of sella turcica.

OPERATION (July 30, 1930).—Right frontal osteoplastic approach. Large pituitary cyst found, containing brown fluid; this was punctured and evacuated by suction.

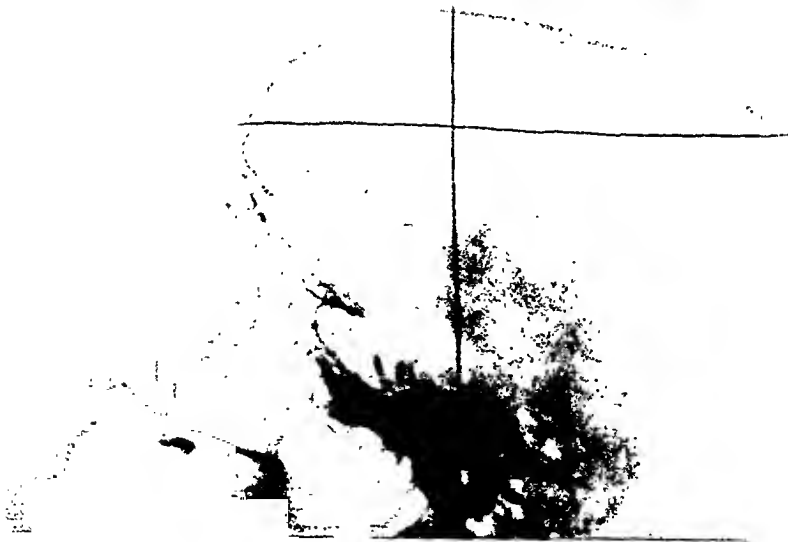


FIG. 391.—*Case 17.* Showing two radon seeds inserted vertically into the sella turcica through a frontal approach.

RADIUM TREATMENT.—Two radon seeds placed in the sella: 1·8 me. each, screened by 0·5 mm. of gold (*Fig. 391*).

AFTER-HISTORY.—The patient is in good health and has returned to his practice with practically full vision.

Case 18.—G. N., male, age 58 years. (Dr. Kinnier Wilson.)

HISTORY.—Two and a half years' history of headache, diplopia, and polyuria. Bitemporal hemianopia; discs pale.

X RAY.—Skiagram shows enlargement of sella turcica.

OPERATION (April 24, 1930).—Frontal route: removal of a great deal of soft tumour.

RADIUM TREATMENT.—Insertion of 3 gold seeds containing 1·4 me. each, screened by 0·5 mm. of gold.

HISTOLOGY.—Adenomatous hyperplasia.

AFTER-HISTORY (November, 1930).—General health good; very occasional diplopia; vision very greatly improved; no headaches, no polyuria.



FIG. 392.—*Case 20.*
Showing two platinum seeds inserted by temporal decompression.

Case 19.—A. M., male, age 18 years. (Dr. Adie.)

HISTORY.—Three weeks' history of vomiting and headache. No fits, weakness of limbs, diplopia, or squint. Bitemporal hemianopia; blurring of upper margin of each disc.

X RAY.—Large pituitary fossa; probably suprasellar tumour.

OPERATION (April 25, 1930).—Right frontal osteoplastic flap: contents of a large suprapituitary partly cystic tumour evacuated.

RADIUM TREATMENT.—Four platinum seeds containing 1·5 me. each, screened by 0·5 mm. of platinum, were inserted into the cavity.

AFTER-HISTORY.—The patient is in good health; has started work as a clerk; vision is very good; symptomless.

Case 20.—E. H., female, age 37 years. (Dr. Birley.)

HISTORY.—Three months' history of headaches, drowsiness, and failure of vision. Marked polyuria. Papilloedema, with +4D swelling and numerous hæmorrhages.

X RAY.—Shows destruction of posterior elinoid processes.

OPERATION (April 24, 1930).—Right temporal decompression. Cystic tumour observed in floor of third ventricle.

RADIUM TREATMENT.—Three radon seeds 1·8 mc. each, screened by 0·5 mm. of platinum, were inserted (*Fig. 392*). Good post-operative recovery.

AFTER-HISTORY.—The patient left hospital free from headache, with papilloedema subsiding, and vision improving.

Case 21.—D. A., female, age 30 years. (From the Surgical Unit, St. Thomas's Hospital.)

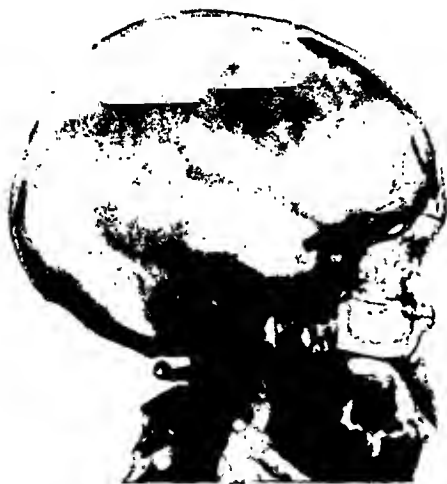


FIG. 393.—*Case 21.* Illustrating silver capsule introduced into the suprapituitary region.



FIG. 394.—*Case 21.* The same. Antero-posterior view.

HISTORY.—Five years' amenorrhœa, polyuria, and failure of vision. No papilloedema. Complete left temporal hemianopia. Enlargement of sella turcica.

OPERATION (Oct. 30, 1930).—Right frontal osteoplastic approach. Suprapituitary tumour exposed, incised, and much of its contents removed.

RADIUM TREATMENT.—One capsule containing 14 mc., screened by 1 mm. of silver, inserted (*Figs. 393, 394*).

HISTOLOGY.—Trabeculae of tumour tissue are separated from one another by very numerous fibrovascular septa. In the intervening tumour tissue the cells have large irregularly-shaped nuclei. Kidney-shaped and sausage-shaped nuclei are frequently seen. Some nuclei stain faintly, others darker. The cytoplasmic matrix is abundant, indefinitely fibrous, and takes a yellowish colour with van Gieson's stain. In this and in the presence of numerous fibrils in a single process it does not quite resemble neuroglial tissue: nor do the characters of the nuclei suggest glioma. It appears to be a form of endothelioma.

AFTER-HISTORY.—Two months later the patient is practically well, with considerable visual improvement. It is a recent case, and is given here to illustrate the substitution of seeds by a capsule.

ANALYSIS OF RESULTS.

From the observation of the cases described and a large number of other cases treated, it seems clear that a certain percentage of patients appear to benefit from radium therapy. Although some cases are completely radio-resistant and do not respond to treatment, others respond rapidly both in general health and in functional recovery. The best results are obtained in astrocytoma and medulloblastoma. It is difficult to assess the value of radium treatment in oligodendrogliomata, as these tumours do well with surgical removal alone and the end-results are not yet available. Spongioblastomata form the most interesting group. Clinically the most malignant, their response to irradiation is variable, some cases having proved successful, others complete failures; but in this group more than in any other post-operative surface irradiation appears indicated, as the survival-rate without radium is as a rule under one year, whilst with radium complete regression of symptoms has been obtained in a few cases.

CONCLUSIONS.

1. The effect of radium on normal brain tissue need not be feared, providing the dose given is within therapeutic range and adequate screening is provided.
2. Interstitial irradiation has a very limited field of usefulness. The effect on the cerebral vascular system, both immediate and remote, must be kept in mind in planning the details of treatment.
3. Surface irradiation by the method described is at present the most useful form of treatment by radium and is of wide application. Maximum dosage over prolonged periods of time appears to be the optimum.
4. The effect of irradiation on the function of the choroid plexuses may prove of practical value, and deserves further study.
5. Decompression is essential for the various reasons stated.
6. Further study is to be directed to the relation between different types of tumour and their response to irradiation.
7. The desirability of repeating treatment even in the absence of symptoms at the end of a certain period of time may have to be considered. And the possibility of combining X-ray therapy with radium treatment offers a new field of investigation.

REFERENCES.

-
- ¹ BAILEY and CUSHING, *Tumours of the Glioma Group*, 1926.
 - ² GREENFIELD, J. G., *Brain*, 1919, xlii, 1.
 - ³ ROSS and CARMICHAEL, *Proc. Roy. Soc. Med. (Neurol. Sect.)*, 1930, May, 27.

*SHORT NOTES OF
RARE OR OBSCURE CASES*

CYST OF THE ILEUM.

By R. Y. AITKEN,

SENIOR SURGEON TO BLACKBURN ROYAL INFIRMARY.

A boy, age 7, was admitted to the Blackburn Royal Infirmary on Feb. 5, 1930. He was brought to the Out-patient Department by his mother, who

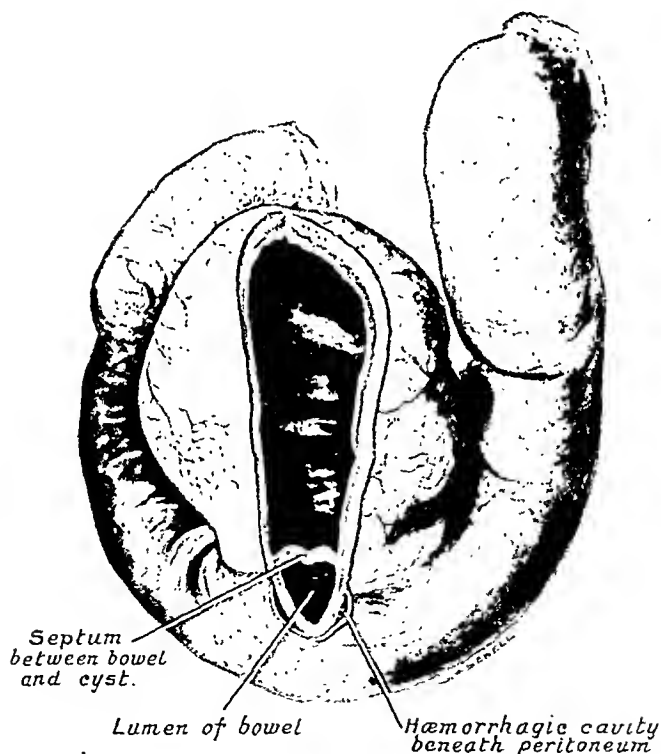


FIG. 395.—Cyst of ileum.

stated that he had been having attacks of abdominal pain and vomiting for the last six months. The medical attendant thought these attacks were due to mild appendicitis. The temperature was normal and the pulse 100.

ON EXAMINATION.—The boy was found to be tender and slightly rigid over the right iliac fossa, and a mass could be felt low down in the fossa.

OPERATION.—The abdomen was opened through a right paramedian incision, and a loop of bowel of a deep purple colour, about nine inches long, was exposed, with a rounded cystic swelling $3\frac{1}{2}$ in. by $2\frac{1}{2}$ in. occupying the mesenteric border of the loop. The discoloration ended abruptly at either

end of the affected gut. The loop and cyst were excised, and an end-to-end anastomosis established. The boy was very ill for several days as the result of acidosis, but ultimately made a good recovery.

The specimen was sent to Sir Arthur Keith, to whom I am much indebted for the report which follows, and for the drawings (Figs. 395, 396). The cyst is of embryological origin—a developmental diverticulum, with the cavity shut off from the lumen of the bowel.

PATHOLOGICAL REPORT.—A loop of ileum which was removed by operation together with an oval cyst measuring 3 in. by $1\frac{1}{2}$ in. which is attached to its mesenteric border. At the site of attachment

the bowel is markedly stenosed. The cyst has a thick muscular wall and is lined with mucous membrane with a villous surface in part of its extent; it has no communication with the lumen of the bowel. In seven inches of the length of the loop of ileum an extensive extravasation of blood has occurred into the wall of the bowel, separating it into an outer and inner layer. The purple discoloration of the peritoneum ceases abruptly near the extremities of the loop.

Microscopical examination of the cyst shows the presence of a thin mucous membrane with ill-developed villi and scanty, ill-developed glands;

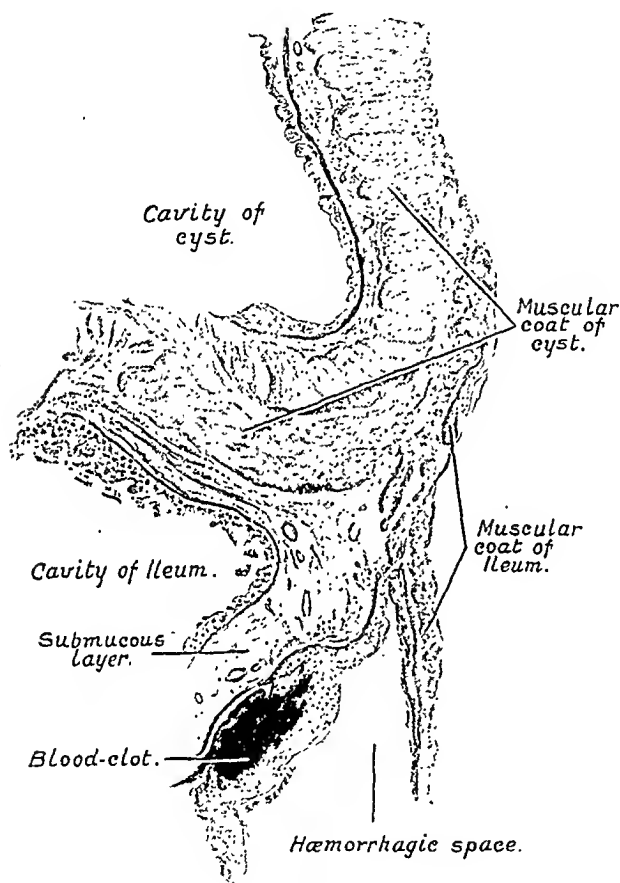


FIG. 396.—Section through the wall of the cyst and adjacent ileum.

the surface epithelium is for the most part desquamated. A well-marked muscularis mucosæ and submucous layer are present, and outside these is a double muscular layer of circular and longitudinal fibres. A section at the site of the union of the cyst with the bowel shows that the septum separating the two does not contain any muscular fibres of the ileum; the muscular coat of the latter can be traced from the bowel over the outer surface of the cyst. The original position of the cyst would thus appear to be in the submucous layer of the bowel.

RETROPERITONEAL CYSTS:

A REPORT OF A CASE OF A LARGE RETROPERITONEAL LYMPHANGIOMA.

By GEORGE D. F. McFADDEN,

ASSISTANT SURGEON TO THE ULSTER HOSPITAL FOR CHILDREN AND WOMEN, BELFAST.

RETROPERITONEAL cysts are of interest both from the clinical and pathological points of view. A cyst is rarely diagnosed, because it is rarely suspected. If the cyst becomes inflamed, the cause of the acute abdomen is as a rule only ascertained when the abdomen is opened. The anatomical relations of the cyst make it difficult to say from what structure it originates, and have led to error in its pathological classification; and the thinness of the cyst wall has not given the pathologist sound material on which to base his opinions.

On account of the absence of pathological proof, there has been much theorizing as to the origin of these cysts. Stoney¹ and Handfield-Jones² both mention as a possible origin the peritoneal cells that should normally disappear during the process of rotation and fixation of the colon. The anatomical relations of some cysts have given rise to the theory that they originate from cells in connection with the urogenital ridge. From its anatomical relations the case reported below might suggest such an origin; yet the piece of tissue taken on the second occasion showed, on microscopical examination, that the cyst was a lymphangioma. The tumour was very like that described as a lymphangioma by Gaudier and Gorse³; the clinical condition was similar to that in the case reported by Stoney in that the patient came under notice with the signs and symptoms of an acute abdomen. When Gaudier and Gorse described their case in 1913 they could find references to only four examples. Of late, however, these cases have aroused a good deal of interest and many excellent papers have been written describing them.^{4, 5} Few observers have been able to identify definitely the origin of the cysts described, so for this reason the case reported below should prove of interest.

CASE REPORT.

HISTORY.—The patient was a boy of 5 years of age when first seen by me. He was brought to the Out-patient Department of the Ulster Hospital for Children, complaining of abdominal pain. The mother stated that he had

been operated upon for a right-sided hernia when 6 weeks old. (I afterwards found that it was a 'hydrocele' that had been removed.) After this operation the abdomen began to enlarge, though the enlargement caused no discomfort. Seven days before being seen by me the boy had an acute pain in the abdomen: at first it was more in the right side, but 'wore over' to the left. Relief was obtained if he got up on his knees and kept his head down on the bed. He vomited several times, was flushed, and sweated. The bowels were regular and there was no disturbance of micturition. The symptoms had been getting worse.

ON EXAMINATION.—The boy was flushed; he lay with his legs drawn up, and had a greatly distended abdomen, the umbilicus protruding like a nipple. There was no venous engorgement or other abnormality of the abdominal wall. Both testes were in the scrotum, and there was no sign of a hydrocele or hernia. The abdomen felt tense, and the boy complained of pain on pressure over the lower abdomen, more marked over the right iliac fossa. No tumour could be felt, and the abdominal distension made detection difficult. The abdomen, except for the epigastrium and a small area below it, was dull on percussion. The temperature was normal, the pulse-rate 120. Nothing abnormal could be felt on rectal examination.

The mother stated that the size of the abdomen had not altered since the onset of the illness and had been the same for four years, causing no discomfort. A satisfactory diagnosis could not be made. All that could be concluded was that the child had developed some acute inflammation superimposed upon a chronic condition. Suggested diagnoses were an acutely inflamed appendix or an inflamed tuberculous gland in a case of tuberculous peritonitis. The symptomless distended abdomen existing for so long was unlike tubercle, and no satisfactory conclusion was arrived at. The child was admitted to hospital.

OPERATION.—Operation next day revealed a large inflamed retroperitoneal cyst that filled the whole of the lower abdomen. The cæum, ascending colon, and small intestine were lifted up beneath the diaphragm. The cyst was horseshoe-shaped. In the middle line it reached about an inch above the bifurcation of the aorta; in the flanks it extended up to both kidney pouches. It reached down into the pelvis behind the mesosigmoid and the mesorectum, but did not touch the bottom of the rectovesical space. The peritoneum over the cyst was red and œdematous. The complete removal of the cyst was considered inadvisable, as it was difficult to strip, and the state of the child would not permit a lengthy operation. The peritoneum was therefore incised and stripped from the upper part of the cyst on the left side, and this part of the tumour was mobilized to bring it down to the anterior abdominal wall in the suprapubic region. The wall of the cyst so mobilized was fixed to the anterior abdominal wall and the redundant part cut off, leaving the cyst to discharge on the surface.

The cyst was multilocular; in one area colonies of small cysts projected outwards, giving the appearance of frog's spawn lying on the wall of the cyst. The fluid in the cyst was turbid, with flakes of lymph scattered through it. A piece of the wall examined under the microscope showed only fibrosis.

The boy made a good recovery from the operation. It was hoped that the inflammation might effect a cure.

SUBSEQUENT HISTORY.—

About a year afterwards the child was admitted with the signs of acute intestinal obstruction. Operation revealed a band at the marsupialized area obstructing the small intestine. This was divided. An examination of the abdomen showed that there was still a moderate-sized retroperitoneal cyst present on the right side. The child made a rapid recovery and the parents were told to bring him back to hospital in three months' time for the removal of the remainder of the tumour.

Three months later the growth was exposed. It was a bluish tense multilocular cyst, and extended from the lower pole of the right kidney and the third part of the duodenum down to the pelvis. A finger-like process extended into the inguinal canal. The cyst was closely adherent to the perinephric fat: the spermatic vein lay anterior to it. With care the whole cyst was completely removed. The child's convalescence was uneventful.



FIG. 398.—To show one narrow lymph-channel with incomplete lining of flat endothelial cells. Adjacent is a blood-vessel containing blood-corpuscles. (*High power.*)



FIG. 397.—A large irregular space is shown into which projects a nodule of lymphocytes showing a 'germ centre'. Plain muscle fibres are seen in the wall at one end. The fatty matrix contains also blood-vessels. (*Low power.*)

The anatomical relations of the cyst as now seen suggested a close relation to the renal structures, and seemed to fit the necessary conditions for one arising from cells of the urogenital ridge. The second pathological report showed it to be otherwise. This report, for which I am indebted to Professor Drennan, shows the cyst to be definitely a lymph-angioma.

The anatomical relations of the cyst as now seen suggested a close relation to the renal structures, and seemed to fit the necessary conditions for one arising from cells of the urogenital ridge. The second pathological report showed it to be otherwise. This report, for which I am indebted to Professor Drennan, shows the cyst to be definitely a lymph-angioma.

PATHOLOGICAL REPORT (Figs. 397, 398).—The matrix consists mostly of fatty tissue, but in places it is fibrous with strands of plain

muscle amongst the collagen fibrils. In this matrix is a sort of sponge-work

of channels, some long and narrow, some in the form of wide spaces. The lining of these is a single layer of flattened endothelial cells—absent in some parts—which are supported by fibrous tissue. Along many spaces are also plain muscle fibres from one to several layers thick. Collections of lymphocytes are seen, sometimes in the walls of spaces, sometimes lying irregularly in the meshes of the matrix, sometimes in the spaces themselves—more especially in the smaller channels. One nodule of lymphoid tissue, having a lymph-node arrangement, is seen in the wall of a space and only separated from the lumen by an endothelial layer. Blood-vessels, both arteries and veins, also occur running through the matrix, but are distinct from the spaces described above. There are scattered small foci of infiltration by polymorph leucocytes—evidently a superadded acute inflammatory reaction. The appearances are those of a cavernous lymphangioma, similar to the congenital 'hygroma' found in the neck and axilla.

Looking over the published papers and the descriptions of cases, this case would suggest that many retroperitoneal cysts are true lymphangiomas, and not cysts arising from local peritoneal cells or from cells derived from the urogenital ridge.

REFERENCES.

- ¹ STONEY, R. A., *Brit. Jour. Surg.*, 1924-5, xii, 789.
- ² HANDFIELD-JONES, R. M., *Ibid.*, 119.
- ³ GAUDIER, H., and GORSE, R., *Presse méd.*, 1913, xxi, 458.
- ⁴ HADLEY, H. M., *Surg. Gyn. and Obst.*, 1916, xxii, 174.
- ⁵ LLOYD, E. I., and HIGGINS, T. T., *Brit. Jour. Surg.*, 1924-5, xii, 95.

CHRONIC SUBLUXATION OF THE DISTAL RADIO-ULNAR ARTICULATION.

BY J. ALLAN BERRY, NAPIER, N.Z.

CHRONIC subluxation of the distal radio-ulnar articulation, or undue mobility of the ulna on the radius, is fortunately not a common condition following injuries about the wrist-joint, and when it does occur is generally so slight that there is very little inconvenience. There is, however, a class of case where it can result in a serious disability, the recognition of which in books dealing with fractures and dislocations is inadequate and the treatment described unsatisfactory. The operation described in the following case—namely, fusion of the inferior radio-ulnar joint, with resection of a portion of the lower quarter of the ulna to allow of pronation and supination—must have occurred to many surgeons, although I have not been able to find any account of it in the literature that is available to me.

C. A. Mc., age 20 years, was admitted to the Napier Hospital on July 17, 1929, suffering from a Colles's fracture of the right radius, associated with a separation of the styloid process of the ulna. Reduction of the fracture

was satisfactorily carried out, and a plaster splint was applied. Although the radius united, the fractured tip of the styloid process did not unite. The patient complained of the undue mobility in an antero-posterior direction of the ulna on the radius. Quite slight pressure on the palm of the hand would cause the head of the ulna to project backwards. As the patient was a carpenter by trade it was impossible for him to use a hammer, and in riding his motor-bicycle the vibration caused the bones to slip on one another, and this caused pain and swelling over the dorsal aspect of the inferior radio-ulnar joint.

OPERATION.—On Feb. 4, 1930, it was decided to fuse the inferior radio-ulnar joint and to resect a portion of the lower quarter of the ulna—the so-called Gallie's operation. This procedure was carried out by means of an incision over the ulna. The inferior radio-ulnar joint was exposed from the dorsal aspect, and its cartilage, which presented a fibrillated appearance, was removed. A bone-peg was then driven through the ulna into the radius. After this about an inch of the ulna in its lower quarter was removed.

SUBSEQUENT PROGRESS.—The wound healed well and the patient in a short time was able to resume his occupation as a carpenter, being able not only to use a hammer without trouble, but also to mix concrete by means of a shovel, which he regarded as a more severe test of the stability of his wrist-joint. At first he complained of the slipping of the lower end of the upper portion of the ulna, but this uncomfortable feeling has now gone. Supination and pronation of the hand are perfect; in fact, the range is rather greater than on the other side. He plays hockey without noticing any disability.

This type of operation would probably be of value in a number of other conditions. In those cases of marked shortening of the radius due to an old Colles's fracture, with great prominence of the lower end of the ulna, leading to pain on rotation of the forearm, it seems likely that the result following an operation such as that described would be better than removal of the prominent end of the ulna. In certain cases of crossed union it would probably be of distinct value in restoring pronation and supination; also in Madelung's deformity the operation should prove useful.

Carson's *Operative Surgery* (p. 87) makes the following statements when dealing with the inferior radio-ulnar joint. "Arthroplasty of this joint has been replaced by Gallie's operation. It is a matter of common experience that fractures of the lower quarter of the ulna have a strong tendency to non-union. Profiting by this, Gallie advised treating ankylosis of this joint by the simple procedure of excising an inch of the lower quarter of the ulnar shaft with the periosteum."

A similar statement is made in the text-book of Rose and Carless. Professor W. E. Gallie in a personal communication states that the operation of removing a segment from the ulna near its lower extremity is not his, and he has not been able to discover how the operation got into the literature under his name, as he has never used the procedure or written about it.

TWO UNUSUAL CASES OF STRANGULATED HERNIA.

By ANDREW FOWLER,

ASSISTANT SURGEON TO THE ROYAL INFIRMARY, ABERDEEN.

Case 1.—The following case of reduction of hernia 'en masse' appears to me to be of sufficient interest to warrant its being placed on record.

HISTORY.—The patient, a male, age 53, was admitted to the Aberdeen Royal Infirmary. He complained of a severe degree of pain in the lower abdomen that had lasted fourteen hours. He had suffered from a 'rupture' on the right side for as long as he could remember. This had been continually increasing in size, but he could always push it back without difficulty; a truss had never been worn. While loading a cart with sheaves about

7 o'clock on the morning of admission the patient felt something suddenly give way in this region. The swelling grew much bigger and could not be pushed back. He persisted in his efforts, however, to reduce the swelling by manual pressure, and after about an hour, to use his own words, made it disappear with great difficulty and much pain. He felt better afterwards and had some food, but a little later felt sick and vomited. He was seen by his doctor, who advised hot applications to the abdomen. He vomited twice later in the day, and his doctor on again seeing him in

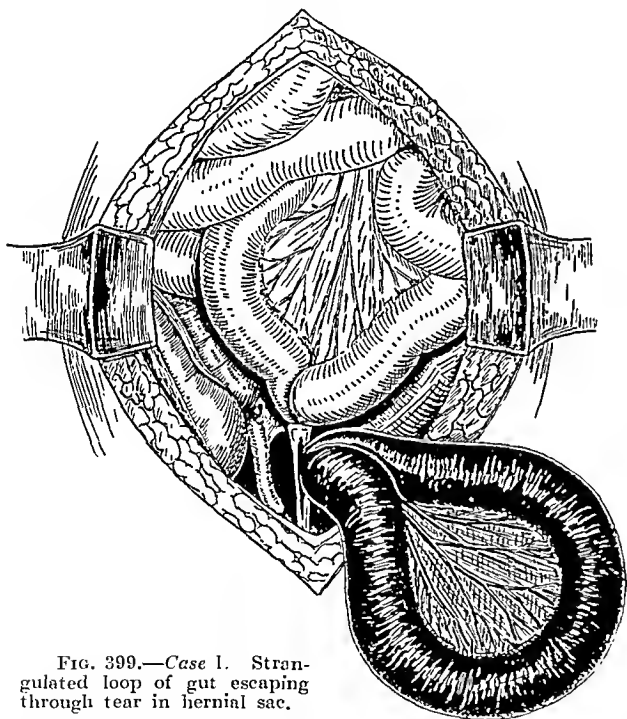


FIG. 399.—*Case 1.* Strangulated loop of gut escaping through tear in hernial sac.

the evening sent him to hospital, having diagnosed strangulated hernia with 'reduction en masse'.

ON ADMISSION.—The patient did not look seriously ill, but was rather cold after an eighteen-mile journey. His pulse was 64, respirations 22, and temperature 97.4° .

ON EXAMINATION.—His tongue was slightly coated all over with a yellowish fur. The abdomen moved fairly well on respiration, but on the right half the movement was definitely less than on the left. No inguinal hernia was visible, but on coughing a small rounded bulging appeared over the

situation of the right internal abdominal ring. On passing a finger along the inguinal canal a firm cord could be felt running obliquely upwards from the outer side of the internal ring, and was lost in the abdominal cavity. On palpating the abdomen a definite area of tenderness was made out in the right lower quadrant, with its maximum intensity slightly to the left of McBurney's point. There was also some tenderness in the epigastrium. Muscular rigidity was also well marked in the right lower quadrant, but in spite of this, a well-defined rounded mass about the size of an orange could be made out, occupying the right iliac fossa. On rectal examination nothing abnormal was made out.

OPERATION.—Operation was decided upon, and under open ether anaesthesia the abdomen was opened by a low right paracentral incision, retracting the rectus muscle outwards. On opening the peritoneum a quantity of blood-stained fluid escaped, and after packing off the small intestine and caecum a black mass was seen in the right upper pelvis. This proved to be a loop of small intestine lying free in the peritoneal cavity. On searching for the site of strangulation this was found to be the neck of the hernial sac which had been forced back into the abdomen. The strangulated loop had escaped through a tear extending through the wall of the sac and parietal peritoneum, as shown in the diagrammatic sketch (*Fig. 399*).

The constricting band was divided, and on examining the sac the double tear of sac and parietal peritoneum was confirmed. The loop of small intestine was wrapped in saline gauze and left for ten minutes, during which time the sac was excised; the internal abdominal ring and the tear in parietal peritoneum were closed

with a double purse-string suture of linen thread. The loop of bowel, about ten inches in length, did not recover sufficiently to justify leaving it, and was therefore resected. End-to-end anastomosis was performed about four feet above the ilcoecal junction. The abdomen was closed in the usual manner without drainage, and the patient made an uninterrupted recovery; he was discharged on the seventeenth day.

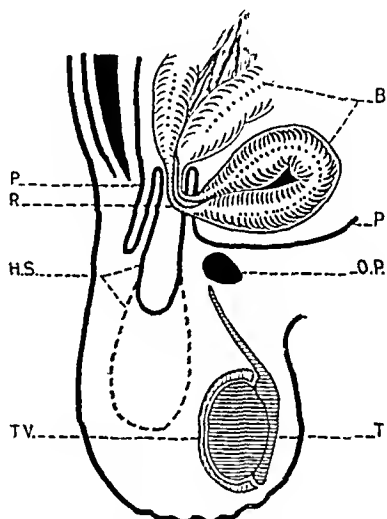


FIG. 400.—Case 1. Condition found at operation. P, Parietal peritoneum; R, Rupture through neck of sac; HS, Hernial sac; TV, Tunica vaginalis; T, Testis; OP, Pubis; B, Bowel.

The difficulty in making an accurate diagnosis in this case was due to the fact that one could hardly credit the man's statement of having reduced the hernia while there was still the appearance of a bubonocoele in the right inguinal canal, and, without the history, the diagnosis of acute appendicitis with abscess might readily have been made on the physical signs. Several accidents of over-zealous taxis are described in recent surgical text-books,

and a specially clear description with diagrams is given in Beattie and Choyce.¹ The conditions found in this case do not seem to correspond to any of the types described therein. I have attempted to reproduce diagrammatically after the manner of the illustrations in the above-mentioned text-book the condition actually found in this case (*Fig. 400*).

Case 2.—The second case is interesting from the history and subsequent findings at operation.

HISTORY.—H. U., female, age 66, was admitted to Aberdeen Royal Infirmary suffering from right-sided abdominal pain and a painful swelling in the right groin. The abdominal pain commenced three days before admission. At first it was generalized—of a dull aching character—accompanied by nausea, but without vomiting. This pain remained during the first day with no disturbance of micturition, and the bowels moved normally. The following day the pain gradually localized itself to the right iliac fossa,

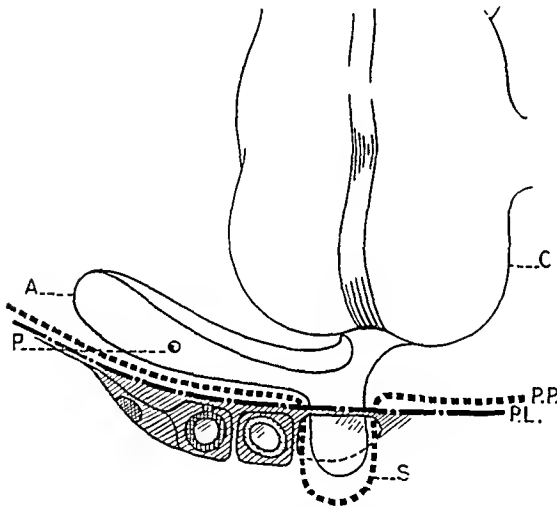


FIG. 401.—*Case 2.* Condition found at operation.
A, Appendix; P, Perforation; S, Sac; P.L., Poupart's ligament; P.P., Parietal peritoneum; C, Cæcum.

increasing in intensity until it became very acute, with occasional bilious vomiting. The bowels moved on this day following an aperient, with no unusual character of the motion. There was no radiation of the pain and no disturbance of urinary function. On the morning of admission the patient complained of a small painful swelling in the right groin which she had not previously observed. A slow increase in the size of the swelling occurred, and the pain in the right iliac fossa continued, but seemed to have some relation to the painful swelling in the groin noted above. On this day the bowels

again moved normally. She was admitted with a diagnosis of strangulated femoral hernia.

ON EXAMINATION.—The pulse-rate was 100 per minute, and the temperature 99°. The patient appeared healthy and well-nourished; the tongue was very furred and the breath foul. The abdomen was symmetrical, and moved fairly well on respiration except in the right lower quadrant. Moderate pressure in the right iliac fossa elicited marked tenderness, which extended upwards to the umbilicus. Marked rigidity of both abdominal recti in the lower halves was apparent. Dullness on percussion was made out in the right flank. There was nothing further of importance to note in the abdominal examination. A rounded swelling about an inch in diameter was observed at the site of a right femoral hernia. This swelling was extremely tender;

it could not be reduced, it was dull on percussion, and there was no impulse on coughing.

OPERATION.—Operation was decided upon immediately, and under open ether anæsthesia a three-inch incision was made over the swelling parallel to Poupart's ligament. A small femoral hernial sac was found after removal of much fat. On opening the sac a tiny piece of what appeared to be gangrenous intestinal wall was visible. This was held gently with a forceps, and the neck of the sac divided with Gimbernat's ligament. The gangrenous portion of intestine on being pulled down was found to be the appendix, the distal two-thirds of which was lying free in the abdominal cavity in a gangrenous and perforated condition. The appendix was removed in the ordinary way by slightly enlarging the neck of the sac, and a fair amount of foul-smelling pus escaped from the femoral hernia opening. The right iliac fossa was drained through a stab incision just inside the anterior superior iliac spine and through the hernial opening. The patient made an uninterrupted recovery. The operation wound healed firmly by granulation, and further operative repair of the crural canal is contemplated at a later date should it prove necessary. From the accompanying diagrammatic sketch (Fig. 401) the condition at operation is seen, and the coloured drawing (Fig. 402) shows the appendix after removal.



FIG. 402.—Case 2. Appearance of appendix after removal.

The interesting features of this case are: (1) The fairly obvious history of an attack of acute appendicitis with the appearance of a very recent strangulated femoral hernia, giving rise to difficulty in deciding which condition was responsible for the majority of the symptoms; (2) The absence of symptoms of intestinal obstruction; (3) The finding of a condition in the appendix very similar to a Richter's hernia of the intestine; (4) That the partial strangulation of the appendix in the manner described above was responsible for the appendicular symptoms.

I am greatly indebted to Dr. F. J. Bowie and Mr. R. Ogg for having prepared the drawings, and to Mr. G. H. Colt for permission to publish the cases.

REFERENCE.

- ¹ BEATTIE and CHOYCE, *System of Surgery*, ii, 657.

REVIEWS AND NOTICES OF BOOKS.

Sensation and the Sensory Pathway. By JOHN S. B. STOPFORD, M.D., F.R.S., Professor of Anatomy, University of Manchester. Demy 8vo. Pp. 148 + xii. Illustrated. 1930. London: Longmans, Green & Co. Ltd. 7s. 6d. net.

PROFESSOR STOPFORD has made a most readable summary of the present state of knowledge of the sensory pathways, with particular reference to the two-stage recovery of sensation after suture of peripheral nerves. The process of regeneration of sensation is carefully described, including the author's own contributions on the recovery of deep sensation. The sensory nervous pathway in spinal cord and brain-stem, and the attributes of thalamic and cortical sensation are outlined.

As so much controversy has centred round the interpretation of the two-stage recovery of sensation, Professor Stopford's view of this is interesting and stimulating. He thinks that the disturbance of intraneural pattern inevitable in regeneration is responsible for the sensory dissociation, and his studies of cases of recovery after nerve compression (without actual severance), and the studies of Sir Edward Schafer on recovery after compression, strongly support this view. Professor Stopford believes that end-organs are specific in function, and that the sensory nerve-fibres (by virtue of their connections) are also specific in function. To explain the phenomenon of reference of sensation occurring in the first stage of recovery, he extends this specialization to the nerve-fibres conveying the elements of second-stage (epieritic) sensation—a procedure which necessitates the postulation of a fibre to appreciate localization ('localization' fibre). To the reviewer it would seem that this postulation of pathways for abstractions of sensation is unnecessary, and that it would be more physiologically sound to regard the whole of second-stage sensation simply as the possibilities for integration which precision in localization gives. The well-authenticated multiple fission of regenerating nerve-fibres might well be used to explain reference and hypersensitiveness, on the basis of more widespread innervation by any one fibre and excess of innervation at any one point. If regeneration of nerve-fibres in heterogeneous channels is responsible for the two stages of recovery of function, it seems that the impossibility of anything approaching accurate microscopical apposition of the ends of nerve-fibres of specific function precludes the attainment of complete functional regeneration.

Congenital Club-Foot (Talipes Equinovarus). By E. P. BROCKMAN, M.Chir., F.R.C.S., Orthopaedic Surgeon, Westminster Hospital, etc. Demy 8vo. Pp. 110 + viii, with 92 illustrations. 1930. Bristol: John Wright & Sons Ltd. 10s. 6d. net.

THIS book is in many respects a model of what a monograph should be. It gives the best account of congenital club-foot that has been published since Walsham and Hughes's *Deformities of the Human Foot*, which appeared in 1898.

The historical survey of the literature divides it into three periods: the first, from ancient times down to the introduction of tenotomy into England by Dr. Little; the second, from that time to the publication of Walsham and Hughes's book, which, as the author points out, is disappointing in that it deals so largely with the minute changes found in the bones of specimens of an advanced or an untreated stage of the deformity; and the third period, from 1900 down to the present time.

With regard to the pathological anatomy, the author's conclusion is that the deformity is due to a displacement of the anterior portion of the os calcis downwards

and inwards beneath the head of the astragalus, and displacement of the scaphoid downwards and inwards until it approximates to, or even touches, the sustentaculum tali or the internal malleolus. By these displacements the head of the astragalus is left very largely uncovered, and only articulates with a limited portion of the socket into which it should enter. The alterations in position of the scaphoid and os calcis with regard to the astragalus are only those which occur in the normal foot when it is adducted, inverted, and plantar flexed, but they are exaggerated in degree. This leads to the view that the principal structural defect is a congenital subluxation of the head of the astragalus within its acetabulum, with shortening of the muscles which control this socket. Emphasis is therefore placed upon the joint displacement and contraction of muscles and ligaments, in opposition to the view that the deformity is primarily bony, which was taken by Walsham.

With regard to the etiology, after a survey of the possibilities of (1) Defect in the germ, (2) Arrest of development, (3) Spasmodic muscular action, and (4) Mechanical causes in utero, the conclusion is that congenital club-foot is caused by a primary failure in development to their full extent of all the tissues of the foot, and that we know nothing of the influence that controls development, or why it should sometimes fail partially or completely.

The methods of treatment described in detail are naturally those which the author himself favours. He is inclined to the use of adhesive strapping as a method of correction in the infant—a method which can undoubtedly have admirable results in the hands of someone who is prepared to devote the time and attention required. The difficulty of this method is that the manipulation and strapping must be repeated at frequent intervals—three times a week is given as ordinarily sufficient in the early stages. The procedure is therefore difficult to carry out except with children who are in hospital or who can be brought up with great regularity; and in private practice it is only possible when a trained orthopædic nurse can be supplied or when the parents can afford very frequent attendances at the orthopædic surgeon's rooms. Manipulation with fixation in plaster-of-Paris is advocated for children whose treatment begins when they are a few months old, but little detail is given as to the exact method and position of fixation. For older untreated or relapsed cases the author describes the method of open correction by division of the structures on the inner side which bind together the scaphoid, the sustentaculum tali, and the internal malleolus until the scaphoid can be abducted and replaced in its proper position in relation to the head of the astragalus. He naturally condemns bone operations, which should only be carried out as a last resort. For choice, if a bone operation is required, he advises an arthrodesis of the tarsal joints, with removal of as much bone as is necessary to correct the deformity.

Chirurgie des Kindersalters. By Prof. Dr. R. DRACHTER, Leiter der chirurgischen Abteilung der Universitätskinderklinik, München; and Dr. J. R. GOSSMAN, Assistenzarzt der Abteilung. Third edition, fully revised and enlarged. Crown 4to. Pp. 1031 + xvi. with 714 illustrations. 1930. Leipzig: F. C. W. Vogel. Paper covers, M. 125; bound in half leather, M. 135.

This text-book of the surgery of childhood is an excellent contribution to a highly important subject. The work is fully inclusive—no matter of importance has been omitted, and in many instances the wealth of detail is almost embarrassing. Professor Drachter contributes the general and introductory matter, and included in this chapter are the recommendations regarding anaesthesia. Close attention is paid to avertin narcosis, and, while views expressed are non-committal, this method has evidently met with favour; in fact it would seem that the dangers of an unduly low blood-pressure are less evident in the child than in the adult.

The section dealing with errors of the mouth, face, and jaw is beautifully and profusely illustrated. The operative methods recommended are those in general use. In cleft palate the Langenbeck-Ferguson procedure is advised, and an appropriate warning is given regarding the risks of forcible alveolar or palate moulding by means of plates or wires. It is regrettable that in this otherwise excellent book

a method of retrograde œsophagoscopy and œsophageal dilatation should be described and figured which is obviously unsuitable to clinical practice and experience. The authors would do well to reconsider this section.

The chapters dealing with abdominal conditions are less detailed than one would have anticipated. Such an important subject as congenital volvulus and gut-rotation errors might have received a fuller consideration; the section on congenital stenosis of the pylorus is also disappointing. Hernia is fully discussed and well illustrated, but the method of applying a dressing to the wound of an inguinal hernia is not so efficient as that employed in this country. The account of the treatment of empyema is critical and satisfying. Rib resections are not favoured, but the value of cannula puncture and suction drainage is stressed. It would have been appropriate to describe the method of 'closed drainage', and fuller recognition might have been given to the work of Graham in relation to the dangers of mediastinal mobilization.

The orthopædic sections of the book are uniformly good with the exception of the account of the pathology and clinical features of acute osteomyelitis. Insufficient attention has been paid to the recent work on the origin and development of the disease.

One of the peculiar values of the book is the inclusion at the end of each chapter of a recent bibliography. It is a plan which enhances the value of a publication, and it might well be adopted more widely.

To sum up—the volume is an excellent presentation, and if some sections are open to criticism, it is because they stand out in contrast to the excellence of the general whole.

Some Aspects of the Cancer Problem. An account of researches into the nature and control of malignant disease commenced in the University of Liverpool in 1905, and continued by the Liverpool Medical Research Organization (formerly the Liverpool Cancer Committee), together with some of the scientific papers that have been published. Edited by W. BLAIR BELL, B.S., M.D. (Lond.), F.R.C.S., Hon. F.A.C.S., Fellow of King's College, London; Director of the Liverpool Medical Research Organization, etc. Imperial 8vo. Pp. 543 + xiv, with 90 plates. 1930. London: Baillière, Tindall & Cox. 63s. net.

THE object in producing this large volume has been to render accessible all published information on the lead treatment of malignant disease and other connected problems. The method adopted has been to reproduce papers published by the members of the Liverpool Medical Research Organization, with minor alterations, and some amplification of recent case-histories, these papers being classified and brought into sequence, while the whole production is cemented by an introduction, short interpolations, and a general summary. The book is, in fact, a reprint on an almost unprecedented scale, and for those who have followed the history of lead treatment in publications and discussions of the last eight years it contains very little that is new. This method of presentation, although admittedly it serves the subsidiary purpose of illustrating the evolution of its subject, is an unsatisfactory one for the reader whose object is to grasp well-ascertained and up-to-date conclusions. The book gives an even more fragmentary impression than might be supposed, owing to the inclusion not only of short papers of lesser importance, but of short excerpts from discussions, many of which are of a purely speculative character. Repetition is necessarily frequent. To have re-written the whole of this work would have been a formidable task, but the condensation so effected, and perhaps the results of the critical review of the whole subject which re-writing would enforce, could certainly have improved its form and enhanced its value.

The need for reconsideration is most evident in the chapter dealing with the actual treatment of malignant disease in the human subject, which is made up chiefly of two articles previously published by Professor Blair Bell, the second being here greatly amplified, and including the histories of about seventy successful cases. The actual number given in a subsequent table as 'believed cured' is 51. Although 566 cases have now been treated, there is no attempt at an analysis of the factors

controlling success or failure. It should, for example, be possible to deduce a relationship between the results of treatment and the type of growth treated. There are theoretical grounds for supposing that the more malignant a tumour is, the more susceptible it will be to the action of lead, and a remarkable number of the successful cases described appear to have been either sarcomas, or carcinomas in comparatively young subjects. This aspect of the question is not even discussed, and no attempt is made to classify results according to the type of growth, or in relation to any other factor which might affect success. Nor are figures given to indicate what proportion of the successful results followed treatment by lead alone and lead combined with X rays: it is surprising that steps should not have been taken to disarm the frequent criticism which has centred on this point. In the series of case-histories there are five categories, one of which is made up of cases treated by lead and X rays combined, but there is no statement that these were the only cases so treated, and at least one other case appearing in a different category is stated to have had X-ray treatment. There is no adequate description of the technique of treatment: the only precise references to dosage and intervals (p. 404) are in an article published in 1926, when experimentation "with different quantities and intervals" was still in progress: the results of this do not appear to be given anywhere later in the book.

These omissions serve to emphasize the unsatisfactory results to be expected from compiling already published papers of different dates, and it is to be hoped that in future editions a critical review of the results achieved may be inserted, with an analysis of results in relation to type and extent of growth, age, quantity of lead given, and nature of any associated treatment. Only by such data can the experience gained in Liverpool be made available elsewhere.

The mode of action of lead on the malignant cell appears still to be imperfectly understood. That a form of coagulation necrosis may be produced has been demonstrated repeatedly (though not consistently), but a detailed histological study at different stages of the process, such as has been made to elucidate the action of radium, still remains to be carried out. The relative concentration of lead in the growth and other tissues forms another question which apparently awaits an answer. The number of analyses of growths and other tissues made is too small, and the results are too variable, to permit of the conclusion that malignant tissue has a special affinity for lead. These are the experimental lines on which progress might well be achieved. When the action of lead is more clearly understood, and when it becomes possible to identify the type of case which will respond to it, the striking success achieved in some of the cases so far treated may well become more common.

The book is cumbersome, but well produced and profusely illustrated. The proceeds of its sale are to be paid into the funds of the Liverpool Medical Research Organization.

Radiologie clinique du Tube digestif. Published under the direction of PIERRE DUVAL, J.-Ch. ROUX, and H. BÉCLÈRE. II. Œsophagus, Intestine, Liver, etc., by J. GATELLIER, Professeur agrégé à la Faculté de Médecine de Paris; F. MOULIN, Chef de Laboratoire à la Faculté de Médecine de Paris; P. PORCHER, Chef de Laboratoire de Radiologie à la Faculté de Médecine de Paris. Fasc. 1 and 2. Royal 4to. Pp. 389, with 823 illustrations. 1930. Paris: Masson et Cie. Bound in two volumes for consignment abroad, Fr. 330.

THESE superb volumes form the second portion of a complete work upon the clinical radiology of the digestive tract. The first section on the stomach and duodenum appeared in 1928, and a large edition was rapidly exhausted. We have no doubt that the present volumes have a similar fate in store. The volumes consist of a series of 416 radiographs showing all portions of the intestinal tract under both normal and pathological conditions. The plates are of the highest order and have been selected with great care, and they are so numerous that it has been possible to show every pathological condition under numerous aspects. Facing each photographic plate is a diagram which shows in outline the particular features for which the plate has been selected, whilst beside the diagram is a brief explanation of what it represents. The diagrams are drawn with very great skill, and represent with

absolute faithfulness the characteristics of the photographs, so that the two together furnish a pictorial clinical pathology of the intestinal tract.

This in itself would be a very considerable achievement, but it is supplemented by a running commentary in which the broad clinical pathology underlying the various conditions is described with a brevity and precision characteristic of the best French scientific literature. Each group of photographs is introduced by a precise description of the technique required in the examination of this particular region. The preparation of the patient is described in accurate detail, and various technical difficulties which may arise are discussed. The various methods devised for rendering this portion of the intestinal tract visible under the X rays are described and criticized, and here the practical details which are introduced are often of incalculable value and will make all the difference between success and failure to those who attempt to follow the procedures advised.

Finally, the most careful details are given of radiographic technique, so that even the most inexperienced can scarcely go astray. It is a pleasure to congratulate our French colleagues on such a brilliant production, as useful to the beginner as to the expert. The former could not make a better investment, for these volumes will save him from many pitfalls; and we venture to think that there is no one so expert but that he will find much to arouse his interest and to assist him in his work. The enthusiast will treasure them as a supreme example of his art and of the ingenuity which can be exercised in its application.

Surgical Diseases of the Thyroid Gland. By E. M. EBERTS, M.D., Surgeon to the Montreal General Hospital; Associate Professor of Surgery, McGill University. With the assistance of R. R. FITZGERALD, M.D., and PHILIP G. SILVER, M.D. Crown 8vo. Pp. 238 + xii, with 48 illustrations. 1929. Philadelphia: Lea & Febiger. \$3.50 net.

THE authors state in the preface that the aim has been to present the subject in a practical way, and within reasonable compass. It may be said at once that they have succeeded admirably. On the clinical side the views are based upon work carried out in recent years in the Goitre Clinic of the Montreal General Hospital by Eberts and his associates. The co-ordination of the work of these associates and assistants has been mutually helpful, and it is obvious how careful the study of the material has been. The terminology of the goitrous thyroid gland is far from being standardized—a survey of the most recent literature will show how diverse personal views still are—so that the observations of a man with the prestige of Eberts, working with capable assistants in a large teaching centre, merit attention. The extent of the material contained within 238 pages is very great. The author has a rare faculty for compression combined with lucidity. The chapters on embryology, anatomy, histology, physiology, and pathological physiology are excellent. The gross and microscopic pathology of the thyroid gland present difficulties to all writers. The author has followed the classification suggested by Williamson and Pearse, in so far as it refers to trophic and plastic lesions, but he does not use this as the basis of his clinical classification. Since some types of thyroid disease are peculiar to certain countries, a uniform classification is naturally difficult. That given is the one which has appeared most suitable for the types of cases met with in the province of Quebec, and Eberts believes it to be generally applicable to the continent of America. The etiology and prophylaxis of simple goitre are well discussed.

The chapters on the subject of Graves' disease are good. The pre-operative and post-operative management given is sound, the risks of operation are clearly set forth, and the case-reports selected to illustrate important points. A chapter is given to the differential diagnosis between Graves' disease and toxic adenoma. A good many writers in different countries believe that the differences are not essential, and are dependent on the extent to which non-toxic pathological change has preceded the onset of the toxic condition. The chapters given to thyroiditis and malignant disease are adequate. The statement that "were examinations less

perfunctory more cases of malignancy would be recognized or suspected before glandular metastases had occurred" is timely, and the necessity for very careful examination of every adenomatous nodule removed is emphasized. Altogether this is a good book.

Lehrbuch der Chirurgie. Dedicated to Professor von EISELSBERG by his pupils. Edited by Professors P. CLAIRMONT (Zürich); W. DENK (Graz); H. von HABERER (Düsseldorf); and E. RANZI (Innsbruck). Royal 8vo. In two volumes. Vol. I. Pp. 869 + xiv, with 389 illustrations. Vol. II. Pp. 658 + xiv, with 298 illustrations. 1930. Vienna: Julius Springer. Paper covers. RM. 66; bound, RM. 69.80.

It must be a proud and happy day for any teacher when he can have such a work as this written, presented, and dedicated to him by his own pupils. It is a comprehensive text-book covering the whole field of general surgery, including a short account of typical operations, but not dealing with the specialities of gynaecology or ophthalmology. The book is on the ordinary lines of an advanced student's text-book, and is well illustrated by line drawings and very clear diagrams. The opening chapter on general surgery is short and concise, covering in forty-four pages the subjects of wounds, inflammation, asepsis, anaesthetics, and the conduct of an operation. There is no special chapter dealing with specific infections—tubercle or syphilis, for example—although of course these diseases are referred to under the affections of special organs.

Röntgenology is dealt with at some length, with an adequate account of deep X-ray therapy. The different organs with their injuries and diseases are described systematically—namely: the blood-vessels, the skull, the central and peripheral nervous system, the face, the jaws, the neck, the nose and larynx, the ear, the mouth and salivary glands, the thorax, the breast, the abdomen, stomach, intestine, appendix, rectum and anus, peritonitis and ileus, the biliary apparatus, liver, pancreas, spleen, hernia, urology (including the male genital organs), the vertebrae, the pelvis, soft parts of the limbs, bones and joints, including fractures and dislocations.

It is hardly possible to appraise or to criticize in any detail a work of this size, but we welcome it as being an authoritative and complete German text-book compiled by leading surgeons who have all received their training in the famous school of Vienna.

Physikalisch-Chemische Probleme in der Chirurgie. By Dr. C. HÄLLER, Privatdozent für Chirurgie in Würzburg. 9" x 6½". Pp. 274, with 62 illustrations. 1930. Berlin: Julius Springer. RM. 19.60.

THE contents of this work may be divided into three parts: (1) The physical chemistry of the body; (2) The application of this to 'general' surgery; and (3) The application to the surgery of special organs. The amount of ground which the author has attempted to cover is enormous, and probably could not be dealt with really satisfactorily in a volume of this size. In consequence, most of the first part will be found very difficult reading by anyone without a fairly extensive previous knowledge of the physical chemistry of the body. The research worker in surgery or surgical pathology will find much material for thought, though he may not regard some of the problems dealt with—for example, that of œdema—as quite so simple as would appear from this book.

The approach to some subjects is different from that usual in this country. For example, in assessing renal efficiency in connection with operability the author carries out as a routine the determination of the freezing-point depression of the blood, the estimation of the 'rest-N', and a refracto-metric estimation of the serum protein.

This book would be useless to the surgeon who had not a proficient knowledge of chemistry.

Lehrbuch der Mund- und Kieferchirurgie. By Prof. Dr. ERICH SONNTAG, A.O. Planmässiger Prof. der Chirurgie und Direktor des Chirurgisch-Poliklinisch Instituts der Universität Leipzig; and Prof. Dr. WOLFGANG ROSENTHAL, A.O. Prof. der Chirurgie an der Universität Leipzig. Royal 8vo. Pp. 444 + xv, with 501 illustrations. 1930. Leipzig: Georg Thieme. Paper covers, M. 24; bound, M. 26.

This volume deals fully with most aspects of surgery of the mouth, jaws, and face, and forms a useful book of reference to the subject for the general surgeon, who for the most part suffers from the disadvantage of meeting with individual cases from this somewhat heterogeneous group at long intervals.

Practical details of local anæsthesia and operations, many of them of a plastic nature, are gone into carefully, and a very large number of excellent illustrations add greatly to the value and interest of the book. The section on epithelioma of the mouth and tongue has not received the attention which its importance would seem to warrant, and no attempt is made to deal with the more recent development of radium therapy in this group of cases. With this exception the book sets out very fully the pathological, clinical, and operative aspects of this interesting group of cases.

The Student's Handbook of Surgical Operations. By Sir FREDERICK TREVES, Bart., G.C.V.O., C.B., LL.D., F.R.C.S., revised by CECIL P. G. WAKELEY, F.R.C.S. (Eng.), F.R.S. (Edin.), Surgeon to King's College Hospital, etc. Fifth edition. Crown 8vo. Pp. 535 + xi, with 190 illustrations. 1930. London: Cassell & Co. Ltd. 10s. 6d. net.

This very solid little book has now reached its fifth edition. The first edition was reprinted no fewer than eight times, the second edition three, and the third edition four times. The book has existed for thirty-eight years—nothing could be more eloquent of the esteem in which it has been held by generations of medical students. Mr. Wakeley has abundantly justified the burden laid upon him in revising a work written by that facile master, Sir Frederick Treves, and nurtured for so many years by Jonathan Hutchinson.

The book contains a detailed account of practically every operation on vessels, nerves, tendons, etc.; in rather less detail it recites operations on bones and joints. Of plastic surgery, mouth and neck surgery, head, spine, and chest operations, there is an adequate account. The genito-urinary system is not forgotten, whilst modern treatment by radium and the cure of varicose veins by injection are carefully described. Operations on the rectum for hæmorrhoids and fistula are treated as fully as such a small book will allow. Abdominal operations and those performed for hernia are all remarkably well described.

The danger in a book of this magnitude is that the author, in a laudable effort to include everything, inevitably makes the book too big—this has been very skilfully avoided; enough has been included, but not too much. Those who are called upon to practice the major abdominal operations would naturally seek much bigger works for their guidance. We who have been brought up on this little book have grown to love it and to be very jealous of its future. We feel it is safe in Mr. Wakeley's hands, and we very strongly recommend the work to our successors. The binding, the paper, the printing, and the quality of the illustrations are excellent.

Die Chirurgie. A System of Surgery. Edited by Profs. M. KIRSCHNER (Tübingen), and O. NORDMANN (Berlin). Fasc. 29 (Vol. II). Royal 8vo. Pp. 1829–2028, with 98 illustrations. 1930. Berlin and Vienna: Urban & Schwarzenberg. RM. 16.

This excellent section on operations on bone and joints by Dr. A. Winkelbauer, of Vienna, completes Volume II, and fills in the gaps to which attention was drawn in our review of Fasciculus 26. The technique of bone suture, bone-grafting, and resection of the long bones is fully described and well illustrated. The approaches to the various long bones are presented rather briefly, and would have gained if accompanied by illustrations showing exact anatomical details. The exposure of the major joints is admirably described, especially the operations on the hip-joint, which contain a wealth of useful information. It is interesting to note the absence

of any reference to the work of Smith-Petersen in adopting and popularizing the method of approach to the hip originated by Sprengel. In removal of the internal semilunar cartilage, division of the internal lateral ligaments is still regarded as permissible, in spite of the growing tendency amongst Austrian and German surgeons to follow British technique in operations for internal derangements of the knee-joint.

The illustrations are bold and well chosen, although many are diagrammatic rather than anatomical. In the comprehensive bibliography British and American authorities are well represented.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Emergency Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London. Volume I. Abdomen and Pelvis. Medium 8vo. Pp. 380 + xx, with 324 illustrations, some of which are in colour. 1930. Bristol: John Wright & Sons Ltd. 25s. net.

A Treatise on Orthopædic Surgery. By ROYAL WHITMAN, M.D., M.R.C.S., F.A.C.S., Consultant to the Hospital for the Ruptured and Crippled, to St. Giles' and St. John's Guild Hospitals, etc. Ninth edition, thoroughly revised, Large 8vo. Pp. 1083 + xii, with 981 engravings. 1930. London: Henry Kimpton. 45s. net.

Diseases of the Chest. By W. J. FENTON, M.D., F.R.C.P., Consulting Physician, Charing Cross Hospital; Physician, Hospital for Consumption and Diseases of the Chest, Brompton, etc., and L. S. T. BURRELL, M.D., F.R.C.P., Physician, Royal Free Hospital, and Hospital for Consumption and Diseases of the Chest, Brompton, etc. Post 8vo. Pp. 384. Illustrated. 1930. London: Jonathan Cape Ltd. 15s. net.

A Text-book of the Surgical Dyspepsias. By A. J. WALTON, M.S., M.B., B.Sc. (Lond), F.R.C.S., Surgeon, London Hospital; Late Hunterian Professor, Royal College of Surgeons. Second edition. Demy 8vo. Pp. 720 + viii, with 286 illustrations in the text and 2 plates. 1930. London: Edward Arnold & Co. 42s. net.

Medizinische Praxis Sammlung für Ärztliche Fortbildung. Edited by L. R. GROTT, A. FROMME, K. WARNEKROS. Volume VIII. Grundzüge der Neurochirurgie. By Prof. Dr. Med. WALTER LEHMANN, Facharzt für Chirurgie in Frankfurt-a.-M. Medium 8vo. Pp. 197 + xii, with 23 illustrations. 1930. Dresden and Leipzig: Theodor Steinkopff. Paper covers, RM. 13.50; bound, RM. 15.

Die Avertinnarkose in der Chirurgie. By Professor D. W. ANSCHÜTZ, Direktor der Chirurg. Klinik, Kiel; Dr. K. SRECHT, Assistent der Chirurg. Klinik, Kiel; and Priv.-Doz. Dr. Fr. TIEMANN, Assistent der Mediz. Klinik, Kiel. Royal 8vo. Pp. 200, with 9 illustrations. 1930. Berlin and Vienna: Julius Springer. RM. 16.50.

Hospital of the Protestant Episcopal Church in Philadelphia. Medical and Surgical Reports of the Episcopal Hospital, 1921-30. Volume VI. Commemorating the Seventy-fifth Year of the Hospital. Demy 8vo. Pp. 460. Illustrated. 1930. Philadelphia: Press of Wm. J. Dorman.

Diagnostics Urgents. Abdomen. By H. MONDOR, Professeur agrégé à la Faculté de Paris; Chirurgien des Hôpitaux. Medium 8vo. Pp. 846 + xiii, with 245 illustrations. 1930. Paris: Masson et Cie. Paper covers, Fr. 125; bound, Fr. 145.

Stepping Stones to Surgery (Anatomy Applied to Surgery). By L. BATHE RAWLING, M.B., B.Ch. (Cantab.), F.R.C.S., Surgeon to St. Bartholomew's Hospital. Demy 8vo. Pp. 228 + xvi, with 97 illustrations. 1930. London: H. K. Lewis & Co. Ltd. 12s. 6d. net.

Technique and Results of Grafting Skin. By H. KENRICK CHRISTIE, M.S. (N.Z.), F.R.C.S., Hon. Surgeon, the Hospital, Wanganui, N.Z. Demy 8vo. Pp. 67 + xii, with 35 illustrations. 1930. London: H. K. Lewis & Co. Ltd. 7s. 6d. net.

Illustrated Primer on Fractures. Prepared by the Co-operative Committee on Fractures. Under the auspices of the Section on Surgery, General and Abdominal, and the Section on Orthopedic Surgery, in co-operation with the Department of Scientific Exhibit, of the American Medical Association. Demy 4to. Pp. 55, with 18 illustrations. 1930. Chicago: American Medical Association. \$1.00.

Diet and Care of the Surgical Case. By REYNOLD H. BOYD, M.D., Ch.B. (N.Z.), F.R.C.S.E., formerly Resident at the Royal Northern Hospital, the Hospital for Sick Children, etc. With an Introduction by C. C. CHOYCE, C.M.G., C.B.E., F.R.C.S., Director Surgical Unit, University College Medical School, etc. Crown 8vo. Pp. 106 + xii. 1930. London: H. K. Lewis & Co. Ltd. 5s. net.

Surgical Emergencies in Practice. By W. H. C. ROMANIS, M.A., M.B., M.Ch. (Cantab.), F.R.C.S. (Eng.), F.R.S. (Edin.), Surgeon to and Lecturer in Surgery at St. Thomas's Hospital, etc.; and PHILIP H. MITCHNER, M.D., M.S. (Lond.), F.R.C.S., Surgeon in charge of Out-patients, Teacher of Operative Surgery, and Demonstrator of Anatomy, St. Thomas's Hospital, etc. Demy 8vo. Pp. 608 + vii, with 158 illustrations. 1931. London: J. & A. Churchill. 18s. net.

Leonardo da Vinci, the Anatomist (1452-1519). By J. PLAYFAIR McMURRICH, Professor of Anatomy, University of Toronto. 7" by 10". Pp. 265 + xx, with 89 plates and illustrations. 1930. London: Baillière, Tindall & Cox. 27s. net.

Deep X-Ray Therapy in Malignant Disease. A Report of an Investigation carried out from 1924-1929 under the direction of the St. Bartholomew's Hospital Cancer Research Committee. By WALTER M. LEVITT, M.B., D.M.R.E., Medical Officer in Charge of the Radiotherapeutic Research Department. With an Introduction by SIR THOMAS HORDER, Bart., K.C.V.O., M.D., F.R.C.P., Chairman of the Cancer Research Committee. Post 8vo. Pp. 128 + xiv, with diagrams. 1930. London: John Murray. 10s. 6d. net.

The Truth about Cancer. With a Foreword by the MARQUIS OF READING. Crown 8vo. Pp. 124 + xv. Illustrated. 1930. Published for the British Empire Cancer Campaign by John Murray, London. 2s. 6d. net.

Der appendicitische Anfall: seine Ätiologie und Pathogenese. By LUDWIG ASCHOFF (Freiburg-i.-Br.); mit einem kurzen Beitrag über die Lymphgefäßverhältnisse am menschlichen Wurmfortsatz by Dr. H. SENG. Medium 8vo. Pp. 125, with 36 illustrations. 1930. Berlin and Vienna: Julius Springer. Paper covers, RM. 12.40; bound, RM. 14.

Die Chirurgie. A System of Surgery. Edited by Profs. M. KIRSCHNER (Tübingen) and O. NORDMANN (Berlin). Fasc. 30 (Index). Royal 8vo. Pp. 219 + vii. 1930. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 12; bound, RM. 14.

THE BRITISH JOURNAL OF SURGERY

VOL. XVIII.

APRIL, 1931.

No. 72.

SOME BYGONE OPERATIONS IN SURGERY.

By SIR D'ARCY POWER, K.B.E., LONDON.

IV. AN HISTORICAL LITHOTOMY: MR. SAMUEL PEPYS.

IF Mr. Samuel Pepys had not been cut for stone on March 26, 1658, it is probable that his *Diary* would have been very different from that which we now read. He had been married for two years, and in the ordinary course of events would have led the domestic life of the period, and would have brought up a numerous progeny. The operation by injuring both his vasa deferentia left him sterile but not impotent, and throughout his *Diary* he shows himself to have been in a state of constant sexual excitement—desirous, but incapable of satisfying his lust with all the pretty but immodest women of his acquaintance.

Unfortunately from a medical point of view the lithotomy was performed two years before he began to write his *Diary*, which begins on Jan. 1, 1659–60, but the day was constantly in his mind, and the entry on March 26, 1660, is a faithful reflection of his thoughts at each return of spring. It runs:—

“This day it is two years since it pleased God that I was cut for stone at Mrs. Turner’s in Salisbury Court. And did resolve while I live to keep it a festival, as I did the last year at my house and for ever to have Mrs. Turner and her company with me.”

Two years later on March 26, 1662–3, the entry is:—

“Up early, this being by God’s blessing the fourth solemn day of my cutting for the stone. At noon came my good guests. I had a pretty dinner for them, viz., a brace of stewed carps, six roasted chickens and a jowl of salmon hot for the first course; a tansy and two neats’ tongues and cheese the second, and were very merry all the afternoon, talking and singing and piping upon the flageolette. We had a man cook to dress the dinner and sent for Jane to help us.”

Mr. Pepys nowhere mentions the name of the surgeon who operated upon him, but we know from other sources that it was Thomas Hollyer, of St. Thomas’s Hospital. Mistress Turner was the wife of John Turner, Serjeant at Law, who lived in Salisbury Court. She was Jane, daughter of John Pepys, of Ashted, and as she lived next door to Pepys’s father—who had a tailor’s

shop in Salisbury Court, Fleet Street—it was convenient to have the operation in her house.

Thomas Hollyer, who operated, was appointed Surgeon for 'Scald heads' at St. Thomas's Hospital in 1638, and on Jan. 25, 1643-4, was chosen Surgeon to the Hospital in place of Edward Molines, Lithotomist to the Hospital. Molines was a Royalist and had been taken in arms against the Parliament at Arundel. He was deprived of his office of Surgeon to the Hospital on the day Hollyer was appointed. After the Restoration Molines was reinstated by order of the king, but as there was no vacancy he became fourth or additional Surgeon.

Hollyer's position as Lithotomist to the Hospital gave him unusual opportunities to perfect his skill in cutting for the stone, as we learn from the Diary of the Rev. John Ward, Vicar of Stratford-on-Avon, who says: "I was at St. Thomas' Hospital on 14th January (1661-2) where the porter told me that Mr. Hollyard cut 30 of ye stone in one year and all lived, and afterwards cut 4 and they all died." It was fortunate for Mr. Pepys as well as for the many generations who have read his *Diary* that he was operated upon before Hollyer and his instruments became septic. It is clear that Hollyer had the entire confidence of Pepys, for he bled him, gave him sound advice on several occasions, and was called in to see Mrs. Pepys when she was ill.

By a curious accident the Sloane Collection of Manuscripts at the British Museum contains two prescriptions written for Pepys on the occasion of his operation. The first is:—

R Pulv. glye. rad.	℥iii	Lactis vaccinæ	
Rad. Althææ	℥ii	Aq. rosarum rub.	lb. iss
Fol. Malv.	℥. * iiiii	Alb. Ovorum No. xv.	
Cinnamomi fract.	℥iss		
Misce ante et distillantur lento igne.			
Sumate hujus ℥vi; Syrup. althææ ℥i½ omni mane et horâ somni quotidie.			
Mr. Peapes.		Dr. J. M.	

* ℥. is the symbol for a handful.

On the next page of the Sloane MS. is a second prescription for a draught to be in readiness when he was cut, and is subscribed, "For Mr. Peapes who was cut for ye stone by Mr. Hollyer, ye 28 March 1658 and had a very great stone taken that day for him." The prescription is signed Dr. J. M.; Dr. G. Jolly. Its chief constituent was lemon-juice, to which a little syrup of radishes was added. The prescription shows that Mr. Pepys was expected to be rather feverish after the operation, but that his physicians did not think that he was in any danger, for it is expressly stated that the mist. alba which it contained was to be 'sine moscho'. In those days musk was considered a sovereign remedy for all who were suffering from collapse or were dangerously ill, and few people died without a viaticum of musk.

The signatures attached to the prescriptions thus accidentally preserved enable us to carry the story of Pepys's operation a little further. The initials J. M. at the foot of each prescription are those of Dr. James Moleynes, the leading lithotomist of his day, the father of Edward Molines mentioned above, and in all probability the master and teacher of Thomas Hollyer. James Moleynes held the office of 'Surgeon for the Stone' both at St. Bartholomew's



Engraved by John Hoyle.

Engraved by E. Colclough.

SAMUEL PEPYS

and at St. Thomas's Hospitals. He had so good a position in the profession that the College of Physicians granted him the unusual privilege of a licence to administer internal medicines in surgical diseases. He signs the first prescription, therefore, alone and with his initials like a physician, for had he not held such a privilege he would, as a surgeon, have signed with his full name. He lived in Warwick Lane, and the minutes of the Court of Governors at St. Bartholomew's Hospital contain the following reference to his appointment as Lithotomist :—

“Vicesimo die Januarii Anno Dei 1622–3.

“It is ordered by the Governors of this Hospitall that James Mullins Chirurgeon in consideracon of his care and paynes which he is to take in cuttinge and cureing of poor diseased persons of the Stone and the griefs and maladies hereunder named, brought to the several hospitals of St. Bartholomew's and St. Thomas' in Southwark shall have the yearly stipend of xxxli. per ann. from XXmas last past so long as he shall discharge the same cures to the good liking of the Governors of the said hospitals, viz. xvl of his said stipend to be paid him quarterly from this hospital and the other xvl from St. Thomas' Hospll. and he is also to be allowed two pounds of towre for every patient for theire more easie and warmer dressings.”

Then follows a statement by Moleynes as to what he undertook to do in return for his thirty pounds :—

“For the annual stipend of thirty pounds I undertake (with God his helpe) my best skill and experience the manuall operation and chirurgical cuer of these issueinge Maladies viz.

“The Cureing by insition the stone in the yarde or Bladder.

“The Cureing of the rupture or falling downe of the Intestines or gutts into the Coddys, by Cuttinge.

“The cureinge by Insition the Carnosity or fleshie substance in the Coddys (i.e., stricture of the urethra as it would now be called).

“Also the curing of Wenns by Insition.

“Desireing that for every patient I may have the allowaunce of 2 pounds of towre for theire easier and warmer dressinge.”

The second prescription is signed by Dr. J[ames] M[ullins] and by Dr. G. Jolly.

Dr. Jolly was George Joyliffe, of Wadham and Pembroke Colleges, Oxford, who graduated B.A. in 1640 and was incorporated M.D. at Cambridge in 1652. He served as a lieutenant in the Royalist army under Lord Hopton, practised at Garlick Hill in London, and died there in November, 1658, a few months after he had attended Mr. Pepys, being barely forty years of age. He was a friend of Glisson, had been one of the discoverers of the lymphatics, and promised to be a distinguished man of science had not death cut short his career.

Pepys's natural curiosity led him to inquire about the operation which he had undergone. Accordingly on Feb. 27, 1662 :—

“Up and to my office, whither several persons came to me about office business. About 11 o'clock Commissioner Pett and I walked to Chirurgeon's Hall (we being all invited thither and promised to dine there); where we were led into the Theatre; and by and by comes the reader, Dr. Tearne,

with the Master and Company, in a very handsome manner; and all being settled, he began his lecture, this being the second upon the kidneys, ureters, &c. which was very fine; and his discourse being ended, we walked into the Hall, and there being great store of company, we had a fine dinner and good learned company, many Doctors of Physique and we used with extraordinary great respect. After dinner Dr. Scarborough took some of his friends and I went along with them, to see the body alone, which we did, which was a lusty fellow, a seaman that was hanged for a robbery. I did touch the body with my bare hand; it felt cold, but methought it was a very unpleasant sight. Thence we went into a private room where I perceive they prepare the bodies, and there were the kidneys, ureters, &c. upon which he read to-day, and Dr. Scarborough upon my desire and the company's did show very clearly the manner of the disease of the stone and the cutting and all other questions that I could think of. . . . How the water comes into the bladder through the three skins or coats just as poor Dr. Jolly has heretofore told me. Thence, with great satisfaction to me, back to the Company, where I heard good discourse, and so to the afternoon Lecture upon the heart and lungs, &c. and that being done we broke up, took leave and back to the office."

The portrait is an etching by C. O. Murray from the painting by John Hayles in the National Portrait Gallery. It was painted in March, 1665—the Plague year. Pepys says of it, "This day I begin to sit and he will make me, I think, a very fine picture and I sit to have it full of shadows and do almost break my neck looking over my shoulder to make the posture for him to work by." On March 30: "And so to Hales's and there sat till almost quite darke upon working my gowne which I hired to be drawn in: an Indian gown, and I do see all the reason to expect a most excellent picture of it." On May 16: "Thence to Mr. Hales and paid him for my picture—£14 for the picture and 25s. for the frame."

THE USE OF LOCAL ANÆSTHESIA IN THE TREATMENT OF FRACTURES, WITH CONCLUSIONS DRAWN FROM FIFTY CASES.*

By JOHN P. HOSFORD, LONDON.

As long ago as 1885 Conway reported four cases in which he had used local anæsthesia while setting fractures. He used 4 per cent cocaine, and reported its use in three cases of Colles's fracture and one of a posterior dislocation at the elbow-joint. In 1903 Reclus reported a case in which he had used local anæsthesia to transport a patient with fractured femur in comfort. Four years later Lerda reported thirty cases in which he had used local anæsthesia, and in the following year Quénu recorded fifteen cases. Braun in 1913 described fifty cases in which either local infiltration at the site of fracture or brachial plexus anæsthesia or other forms of conduction anæsthesia were used. In 1921 Fulton in London described in a short article five cases where he had used 1 per cent novocain in setting fractures, and he had good results with its use in all except one. Since then a number of articles have appeared in the foreign literature recording its successful use. Riee describes in some detail the technique for local anæsthesia in cases of Colles's fracture by infiltration with 1 per cent novocain around the site of fracture. It has been used in Böhler's fracture clinic in Vienna in some thousands of cases with success. He uses 2 per cent novocain and injects it directly into the site of fracture.

Various Uses of Local Anæsthesia.—There are several different purposes for which local anæsthesia may be employed in treating fractures. Foremost amongst these is its use for relieving pain and abolishing muscle spasm while a fracture of almost any bone is being reduced and splinted. In fractured femur cases it can be used to overcome pain while the apparatus for skin or skeletal traction and the necessary splint are being put into position and applied to the leg. For skeletal traction either a caliper may be applied or a pin may be passed painlessly through the bone under the effect of local anæsthesia. For obtaining traction in difficult cases of fractured phalanges novocain anæsthesia may be used for passing a sharp steel wire through the pulp of the finger. It may also be used with considerable advantage in cases where a patient with a fracture of a large bone, especially the femur, has to be transported either a short or a long distance, as the shock and mental distress from the pain and muscle spasm brought on by the almost unavoidable jolting associated with the journey can be greatly alleviated. It must, of course, be remembered that during transport the limb should be securely splinted, lest, with the pain abolished, a patient may be tempted to move the

* From the Surgical Professorial Unit, St. Bartholomew's Hospital.

limb and so do serious damage thereto. In the present series of cases local anæsthesia has been used for all these purposes.

Methods of Using Novocain in Fractures.—

1. The whole area around the fracture may be infiltrated with novocain introduced through several punctures on different sides of the limb.

2. Novocain is injected directly into the hæmatoma at the site of the fracture between the bone fragments. A hæmatoma is always present and extends to all parts of the fracture; thus the anæsthetic solution will reach all the fractured surfaces of bone. If one bone is fractured in two or more places, or if more than one bone is fractured, then it will be necessary to make more than one injection.

3. By injecting suitable peripheral nerves on the proximal side of a fracture in a limb it is possible to relieve pain and muscle spasm at the site of the fracture.

4. In open fractures, or in otherwise difficult fractures in the upper limb, anæsthesia may be obtained by injecting novocain into the brachial plexus.

5. In the lower extremity, with similarly complicated cases, spinal anæsthesia may be used.

Advantages of Local Anæsthesia.—The use of local anæsthesia has many advantages over general anæsthesia. The most obvious, though by no means the most important, is its use in those patients who, on account of age or for other reasons, are unfit for a general anæsthetic; and it cannot be too strongly emphasized that for setting and splinting fractures properly it is almost invariably necessary to have an anæsthetic much deeper and more prolonged than 'a whiff of gas'. When working alone, local anæsthesia overcomes the difficulty of being without an anæsthetist. Some patients who absolutely refuse to have a general anæsthetic will agree to local anæsthesia. In severely shocked patients a fracture may be safely 'put up' at an earlier date than if a general anæsthetic has to be used.

More often than not the advantages already given (though they are obvious ones) will not appear in the majority of cases. The following advantages, however, are very real, and are present in many instances.

While reducing the fracture and reviewing the correct position and applying splintage the surgeon has the co-operation of the patient. For example, in the case of a severe eversion fracture-dislocation in the region of the ankle-joint it is convenient to have the patient sitting on a high table with the legs hanging over the end, and after reduction is apparently obtained its completeness or otherwise can be well determined by asking the patient to move his ankle while at the same time the surgeon holds the malleoli firmly between his hands. Only if reduction is satisfactory will these active movements on the part of the patient appear normal. It is also extremely instructive, as well as helpful, in the case of a Colles's fracture to see the improvement in the range of active movement which occurs as the fragments are brought into the correct position. This effect, of course, is entirely lost with a patient under general anæsthesia, since passive movements do not truly represent active movements. The advantage of the patient's co-operation and consciousness with local anæsthesia is also very evident in cases of fracture at the upper end

of the humerus, where it is necessary to apply some form of shoulder abduction splint other than plaster, because the only thing more difficult than fitting such a splint to an unconscious person lying down is fitting it to a patient who is being held in a sitting position while under the influence of a general anæsthetic. With the patient sitting down and freed from pain and muscle spasm by local anæsthesia, the reduction of a difficult fracture at the upper end of the humerus is as easy as it ever is, and the application of the splint is perfectly simple.

For patients who are going to proceed home immediately after the reduction of the fracture, local anæsthesia has a great advantage, as it is rare for nitrous oxide to be a really efficient anæsthesia for the proper reduction and splinting of a fracture.

Similarly, the immediate X-raying of a patient after reducing a fracture under local anæsthesia, instead of perhaps waiting until the following day, when he will have recovered from a general anæsthetic, is a great advantage. This is actually one of the greatest advantages, because if on the immediate development of the skiagram the position of the fragments is seen to be unsatisfactory, the splints may be removed, the position of the fragments altered, and re-splinting done without any fresh anæsthetic being required. With general anæsthesia there is sometimes a tendency to allow an only moderate position to pass owing to the patient's objection to having another anæsthetic so soon, or to the difficulties of obtaining an anæsthetist.

On the few occasions when a surgeon wishes to set a fracture under the X-ray screen local anæsthesia has most obvious advantages.

Disadvantages and Contra-indications.—In very young children local anæsthesia for setting a fracture is clearly not convenient. The youngest child in this series of cases was 7 years old, and she neither cried nor complained while the position of her fractured tibia was corrected under local anæsthesia.

In cases where it is impossible or very inconvenient to obtain a skiagram when a fracture is first seen, general anæsthesia is more certain to give good results than local anæsthesia, except in the case of typical classical fractures, because without skiagrams the general alinement of fragments can in most cases be well obtained under general anæsthesia; whereas without skiagrams, when much swelling is present, and especially where there is more than one fracture, it is difficult to inject novocain exactly at the site of fracture.

The time taken when using local anæsthesia is a little longer than in the case of general anæsthesia.

Sometimes it is not possible to obtain novocain at short notice and there is a slight amount of trouble involved in preparing it, but if a small stock of solid novocain is kept, a solution is easily and quickly prepared (*see below*).

In cases of open fractures local injection at the site of the fracture is contra-indicated. In such cases either general anæsthesia or brachial-plexus block or spinal anæsthesia may be used. With an extensive injury of the lower extremity it may be extremely difficult and painful to the patient to roll him on his side or sit him up to perform lumbar puncture, and so general anæsthesia must of necessity be used.

Risk of introducing infection by injecting novocain into fractures might

be advanced as a contra-indication to its general use, but with ordinary care this should not arise. In no single case in this series was there the slightest evidence of infection. In Böhler's fracture clinic, where novocain has been used in a very large number of cases to the complete exclusion of general anæsthesia, it is claimed that no case of infection has occurred.

CASE REPORTS.

During the last year local anæsthesia has been used in the treatment of fractures in the Surgical Professorial Unit at St. Bartholomew's Hospital on many occasions. Excellent results have been obtained with its use in fractures of nearly all the long bones. It has been used in fractures of the following bones :—

<i>Clavicle</i>	<i>Femur</i>
<i>Humerus</i> —	<i>Tibia</i> —
(a) Surgical neck	(a) External condyle
(b) Shaft	(b) Shaft
(c) Supracondylar fracture	<i>Fibula</i> —
<i>Radius</i> —	External malleolus
Lower end (many different degrees and varieties, including separation of the distal epiphysis).	<i>Tibia and fibula</i> —
<i>Carpal scaphoid</i>	(a) Fractures of shafts of both bones
<i>Metacarpals</i>	(b) Fracture-dislocations of ankle-joint, involving tibia and fibula. (Many degrees and varieties.)
<i>Phalanges</i> —	
Multiple fractures with lacerated hand	

It has also been used for introducing Steinmann's pins for obtaining skeletal traction in cases of fractured femur and tibia. The pin has been put through the tuberosity of the tibia in the former case and through the os calcis in the latter.

The following 10 cases out of the series of 50 will serve to show the use of local anæsthesia in treating various types of fractures.

Case 1.—Male, age 34. City police constable. Fractured shaft of left humerus.

Considerable angulation and much pain. Nineteen hours after fracture 50 c.c. of 2 per cent novocain were injected, half the quantity from the posterior and half from the anterior aspect. In about five minutes the temporary splint with which the patient had arrived was removed and the angulation at the site of fracture corrected by manual traction. This was done easily and without causing pain, whereas previously the slightest movement had caused intense pain. The arm was put in a Thomas arm splint with fixed extension. *Fig. 403* shows the fracture as seen immediately after this reduction. The shortening had been very easily overcome, as the muscle spasm while putting up the fracture had been abolished. The patient returned to duty in eleven weeks.

Case 2.—Female, age 21. Typist. Colles's fracture of left wrist.

Fig. 404 shows the extremely severe displacement: 20 c.c. of 2 per cent novocain solution were injected dorsally and 10 c.c. antero-laterally five hours after the accident. Perfect anæsthesia and muscle relaxation were obtained while the fracture was reduced. *Fig. 405* shows the position after the reduction. The patient was in tears and extremely nervous before the injection was begun, but after the reduction voluntarily admitted that it had not hurt 'the slightest bit', and proceeded immediately to the X-ray department to have the position checked.



FIG. 403.—*Case 1.* Fracture of humerus reduced under local anesthesia.

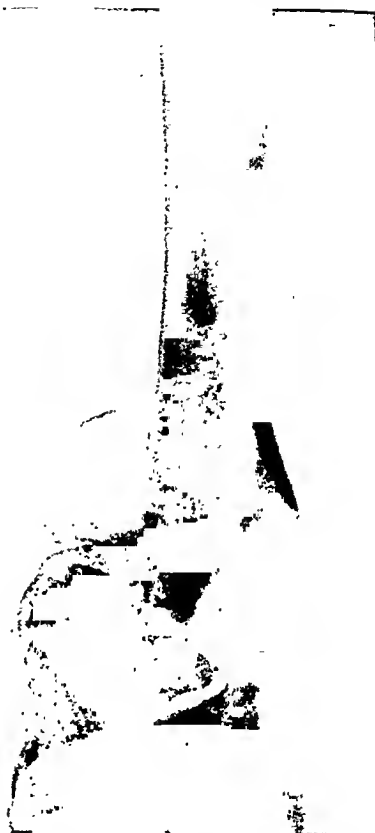


FIG. 404.—*Case 2.* Colles's fracture before reduction. Extreme backward tilting as well as backward displacement.



FIG. 405.—*Case 2.* After reduction, with dorsal plaster splint in position.



FIG. 406.—The position of the needle while injecting a Colles's fracture.

Fig. 406 shows the best angle at which to inject the solution from the dorsal surface, and the position, with relation to the fragments, at which one aims at putting the solution. In *Fig. 407* the line indicates the site of a Colles's fracture, and the needle is put in about three-quarters of an inch, more or less (depending on the amount of fat and swelling present), proximal to the fracture.

Case 3.—Male, age 9. Displaced distal radial epiphysis.

There was considerable dorsal displacement of the epiphysis: 15 c.c. of 2 per cent novocain were injected dorsally, and perfect anæsthesia and reduction were obtained. The recovery was uneventful, and the result perfect.

Case 4.—Male, age 29. Fractured carpal scaphoid.

There was a fracture of the ordinary 'snapped-waist' variety with some pulverizing of bone, with the latero-distal fragment somewhat rotated. There was much pain and muscle spasm, so that it was impossible to dorsiflex the wrist. About nine hours after the accident 15 c.c. of 2 per cent novocain were injected to the site of fracture by way of the anatomical snuff-box: at the same time the hand was deviated towards the ulnar side somewhat. The relief from pain and muscle spasm, though not complete, was then sufficient to allow the hand to be dorsiflexed to a satisfactory position, and it was maintained by a plaster cast.

Case 5.—Female, age 22. Fractured femur.

There was an almost transverse fracture of the middle of the shaft of the femur: 60 c.c. of 2 per cent novocain were injected at the site of fracture and 10 c.c. at each side of the tuberosity of the tibia. A Steinmann's pin was then passed through the tibial tuberosity, and a weight attached to it by a cord over a pulley, after the leg had been adjusted in a Thomas splint. The movements necessarily associated with these procedures caused no pain at all in the femur, and the sight of the pin through her tibia surprised the patient, as she had not felt the slightest pain while it was being hammered in, although she felt the hammering.

Often the periosteum is slightly sensitive, though the other tissues may have been well anæsthetized.

Case 6.—Male, age 50. Licensed victualler. Fracture-dislocation of ankle-joint.

Fig. 408 shows the very severe backward displacement, and the position of the fibula shows the great rotation. There was also great eversion. On the same day as the accident 30 c.c. of 2 per cent novocain were injected antero-internally below the projecting lower end of the tibia, and 10 c.c. antero-externally. These injections went into the ankle-joint, and perfect anæsthesia and abolition of muscle spasm were obtained so that reduction was easy. The patient then carried out active movements, and crepitus was felt as the foot was held in position, but this

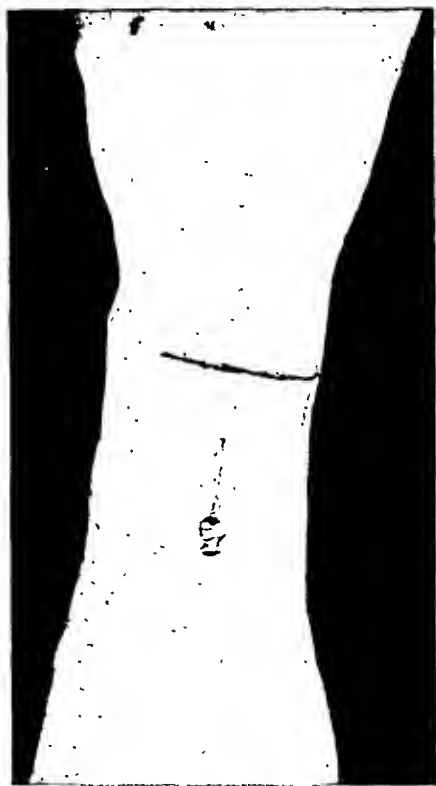


FIG. 407.—Dorsum of forearm and hand. The level of a Colles's fracture is indicated, and the site for injection shown.

caused no pain. Good position was obtained, and in seventeen weeks the patient was discharged from treatment with a good ankle.

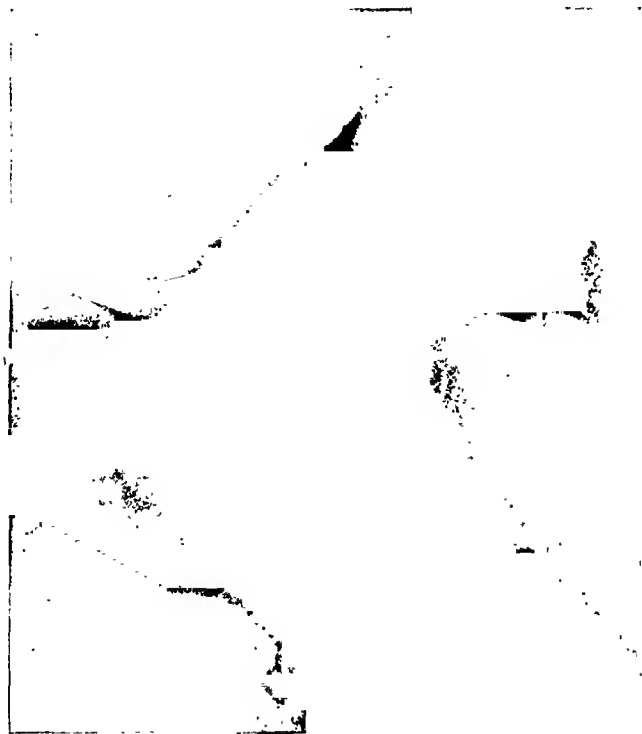


FIG. 408.—Case 6. Pott's fracture-dislocation at the ankle-joint.

Case 7.—Male, age 43. Carman. Lateral dislocation at ankle.

Fig. 409 shows the unusual dislocation at the ankle. The astragalus with the fibula has been displaced laterally away from the tibia, but there was no fracture except at the very upper end of the fibula: 52 c.c. of 2 per cent novocain were injected on the internal aspect immediately anterior to the internal malleolus. Apart from the first needle prick the patient experienced no pain at all. Perfect position was obtained.

Case 8.—Female, age 61. Pott's fracture.

Fig. 410 shows the fracture, and the considerable lateral displacement: 25 c.c. of 2 per cent novocain were injected into the comminuted fracture of the fibula and 25 c.c. antero-internally, in the region of the internal malleolus, into the ankle-joint. Extremely large fracture blisters were present at the time of injection. No pain was felt at the time of reduction or during the application of the plaster. The patient obtained excellent function, and the position was good.

Case 9.—Male, age 52. Fractured tibia. Steinmann's pin in os calcis.

There was an oblique fracture at the junction of the middle and lower thirds of the tibia, with considerable shortening. A week after the accident a Steinmann's pin was passed through the os calcis after injecting 10 c.c. of 2 per cent novocain solution at each side. There was slight pain only as the pin penetrated the periosteum.

Case 10.—Male, age 18. Lacerated hand with multiple fractures.

Brachial-plexus anæsthesia after the method of Kulenkampff was used, as there were extensive lacerations of the palm and fingers, and fractures of phalanges in the thumb, index, and middle finger. Ten c.c. of 2 per cent novocain were injected into the plexus above the clavicle, and a further 10 c.c. around it. Good anæsthesia was obtained in the hand, except over the little finger and ulnar side of the hand; this part, however, was not injured.



FIG. 409.—Case 7. Lateral dislocation at ankle-joint with tearing of interosseous ligament between tibia and fibula.

FIG. 410.—Case 8. Eversion fracture-dislocation at the ankle-joint before reduction. There was no backward displacement at the ankle.

Local Anæsthetic Used.—In all except two cases a 2 per cent solution of novocain in normal saline has been used. A 0.5 per cent solution was used in the case of two Colles's fractures, but the results obtained were not so satisfactory as with the stronger solution.

The novocain solution is always freshly prepared by boiling either 50 c.c. or 100 c.c. of normal saline in a flask. It is then removed from the gas-ring, and either 1 or 2 gm. of solid novocain is added and the solution brought to the boil again only for a few seconds. Adrenalin has not been used; it is apparently not necessary, because the novocain is injected directly into the hæmatoma and there is not the rapid absorption and removal of the novocain that occurs when it is injected into normal tissues.

The quantities injected have varied from 10 to 144 c.c. The latter quantity was used in the case of a patient who had fractured her femur, tibia,

and fibula, and in addition to injecting fractures in these bones injections of novocain were also made on both sides of the tuberosity of the tibia for the introduction of a Steinmann's pin. Although the quantity injected in this case at one sitting was 144 c.c. there were no apparent toxic effects, nor have there been in any case in this series.

Technique of Injection.—In this series the method employed has been that of injecting the novocain solution directly into the hæmatoma. The site of injury is X-rayed, and, the fracture or fractures having been exactly located, the injection is made so that the needle point will go as close as possible to the fractured ends. The skin is sterilized with ether only, and with the finest hypodermic needle a wheal is raised. Then a 0.8-mm. needle is passed directly down to the fracture; blood will now sometimes flow back through the needle, and this means that the hæmatoma has been reached; usually, however, there is not sufficient pressure in the hæmatoma for this to occur. A 10-c.c. or 20-c.c. syringe charged with the novocain solution is now attached to the needle and 5 c.c. are injected. The piston of the syringe is then withdrawn a little, and if blood-stained fluid returns into the barrel, it is known that the hæmatoma has been reached. If blood-stained fluid cannot be withdrawn, the position of the needle must be altered until the hæmatoma is found, and then the injection is made. One can usually tell before actually withdrawing the piston whether the needle is in the hæmatoma or not, as during the injection of the 5 c.c. the piston is very easily pushed down if the hæmatoma is being injected; whereas if the injection is being made directly against normal bone where there is no hæmatoma, the piston has to be forced down fairly hard. However, although the piston may apparently be easily pushed home, the precaution of first withdrawing it and seeing the blood-stained fluid should not be overlooked.

No rule can be given as to the quantity it is necessary to inject, but about 20 c.c. has usually been used for Colles's fractures, 40 to 50 c.c. for Pott's fractures, and 20 to 40 c.c. for fractures of the shaft of the tibia, depending upon whether they are comminuted or not. The needle is then removed and gentle pressure kept on the site of puncture for about five minutes. A piece of sterile gauze is laid over the area and manipulation is carried out, as by this time anæsthesia is usually satisfactory and muscle spasm abolished. In a few cases where anæsthesia has not been complete a further injection has been made, approaching the fracture from a different direction, as usually the primary failure is due, not to an insufficient quantity of solution having been injected, but to its not having reached both sides of the fracture, owing, perhaps, to impaction of the fragments. In most cases, except for the prick of the first needle, no pain is caused by the injection, though sometimes there is a slight sickening pain as the needle encounters the periosteum. As regards the length of time after the fracture at which anæsthesia can be obtained, no limit can be given from this series, as the longest period after the fracture that the injection was made was five days, and then good anæsthesia was obtained.

Results of the Use of Local Anæsthesia.—In only one case was there a complete failure to obtain relief of pain and muscle spasm, so that general anæsthesia had to be resorted to. This was a case of fracture of the lower

end of the tibia and fibula without displacement. There was a considerable amount of swelling, so that it was not possible to palpate the fragments, and a skiagram had not been taken. Failure here was obviously due to mistaking the level of the fracture in the tibia, as it was actually two inches higher than was thought at the time. In another case, that of fractures of the second and third metacarpals, where skiagrams had not been previously taken, there was no relief from pain after the injection, but the correction of the angulation was proceeded with and a satisfactory position obtained. In a third case, which was done at night almost immediately after the fracture had occurred (no skiagrams had been taken), there was not complete relief of pain, because what was thought to be a single oblique fracture of the shaft of the tibia was actually a double one and only the lower one had been injected; however, though there was not complete relief from pain at the manipulation, the patient did not complain while a plaster cast was being applied, and the rigid protective spasm which was present had disappeared. Perfect position was obtained. Thus, out of about fifty cases only three may be said to have been failures when using 2 per cent novocain. In the other cases it has been quite surprising to see what perfect relief from pain and excellent abolition of muscle spasm has been obtained. The patients themselves have frequently remarked that they have felt the bone slip back into place, but that it does not hurt, and then, when a full range of active movements has been carried out and crepitus can be felt, the patient has watched it uncomplaining and with interest.

In cases where 'fracture blisters' have been present at the time of injection these have been avoided with the needle; and, especially in one case, it was interesting to note how the blister increased in size as the injection of novocain was made into the fracture at some distance from the blister.

There has been no case in which there has been any evidence of infection having been carried into the fracture, nor any instance of toxic effect having occurred from the novocain.

CONCLUSIONS.

1. Local anæsthesia for setting fractures, when performed by injecting 2 per cent novocain directly into the hæmatoma which surrounds all parts of the fracture, is a safe and practicable procedure.

2. Beyond the prick of the first hypodermic needle there should be no pain, except occasionally as the needle penetrates the periosteum, and sometimes as the bruised soft tissues are pressed upon during reduction. The latter, however, only occurs in fractures due to direct violence.

3. Muscular relaxation should be as good as it is with general ether anæsthesia, and better than is usually obtained with nitrous oxide.

4. Skiagrams should always be taken before injecting the novocain so as to be as certain as possible of getting the solution between, or against, the fragments, as failure to do this is almost invariably the cause of failure to bring about relief of pain and relaxation of muscle spasm, provided a freshly prepared 2 per cent solution of novocain is used.

BIBLIOGRAPHY.

- BÖHLER, LORENZ, *Treatment of Fractures*, 1929.
 BRAUN, H., *Deut. med. Woch.*, 1913, xxxix, 17.
 COHEN, IRA, *Jour. Amer. Med. Assoc.*, 1926, lxxxvi, 1896.
 CONWAY, N. Y. *Med. Jour.*, 1885.
 CUMSTON, C. G., *Internat. Clinics*, 1918, iv, 213.
 DOLLINGER, B., *Zeits. f. Chir.*, 1913, xl, 763.
 FORRESTER, *Amer. Jour. Surg.*, 1928, v, 296.
 FROSTELL, G., *Zeits. f. Chir.*, 1925, lii, 1308.
 FULTON, R. A. H., *Brit. Med. Jour.*, 1921, ii, 790.
 HAGENBACH, E., *Schweiz. med. Woch.*, 1921, li, 488.
 LERDA, G., *Bull. et Mém. Soc. de Chir.*, 1908, xxiv, 976.
 MORA and WILLIS, *Amer. Jour. Surg.*, 1930, viii, 1062.
 RECLUS, *L'Anesthésie localisée par la Cocaine*, 1903, 266. Paris.
 RICE, CARL O., *Jour. Amer. Med. Assoc.*, 1928, xcix, 1768.
 SCHNEK, F., *Beitr. z. klin. Chir.*, 1928, 484.

THE VASCULAR COMPLICATIONS OF CERVICAL RIB.*

By E. D. TELFORD,

PROFESSOR OF SYSTEMATIC SURGERY, UNIVERSITY OF MANCHESTER;

AND JOHN S. B. STOPFORD,

PROFESSOR OF ANATOMY, UNIVERSITY OF MANCHESTER.

THE existence of a cervical rib may lead to symptoms which are predominantly sensory, motor, or vascular. The sensory and motor phenomena have been described on many occasions and there is general agreement as to their main features. The vascular symptoms are, however, much rarer, and still present many points of difficulty in their interpretation.

The following study is based on personal experience of three cases of cervical rib in which the prominent symptoms were due to thrombotic obliteration of the arteries of the arm. A short account of the three cases follows:—

Case 1.—E. S., male, age 57, was first seen on Nov. 9, 1929.

HISTORY.—The patient had been a vanman all his life, driving horses till 1915, and after that date handling a motor lorry. He had had no illness and had never lost a day's work, but twelve months before admission to hospital he noticed that the fingers of his right hand, more especially the index, were white and painful. He continued at his work, which involved handling heavy packages, until Easter, when, his hand being much worse and an ulcer appearing at the base of the nail of the index finger, he was obliged to give up. The hand became weaker and the pain worse so that he lost much sleep.

ON ADMISSION.—The condition of the right hand was as shown in *Fig. 411*. The whole index finger was in a state of dry gangrene and the distal halves of the remaining fingers were all in a condition of impending gangrene. No arterial pulse could be felt anywhere in the arm below a point one inch higher than the lower border of the pectoralis major tendon. Above this point there was a very distinct and thumping pulse—the third stage of the subclavian artery pulsating visibly. A routine examination of the pulse throughout the rest of the body showed that it was everywhere normal.

There was no wasting of the small muscles of the hand, and sensation to cotton-wool and pin-prick was normal everywhere excepting over the gangrenous areas. The patient was a thin man obviously worn by pain and loss of sleep. The Wassermann reaction had been done before admission: it was reported negative, and was repeated after admission with a like result.

OPERATION (Nov. 11).—A portion of the cervical rib was excised. The subclavian artery and the lowest trunk of the brachial plexus were dissected very deliberately and with great care so that their relations to the rib might be observed with the least possible disturbance. It was apparent that the subclavian artery was in no way embarrassed; the vessel passed freely over the rib with a good clearance and no mark of any pressure. The lowest trunk of the plexus was lying

* From the Departments of Surgery and Anatomy, University of Manchester.

hard on the rib in the notch shown in the photograph of the excised bone (*Fig. 412*). The removal of the rib was quite easy in spite of the fact that the more simple subperiosteal resection was not done; in fact, it ought never to be done, since to leave the periosteum is to risk a recurrence of the rib.

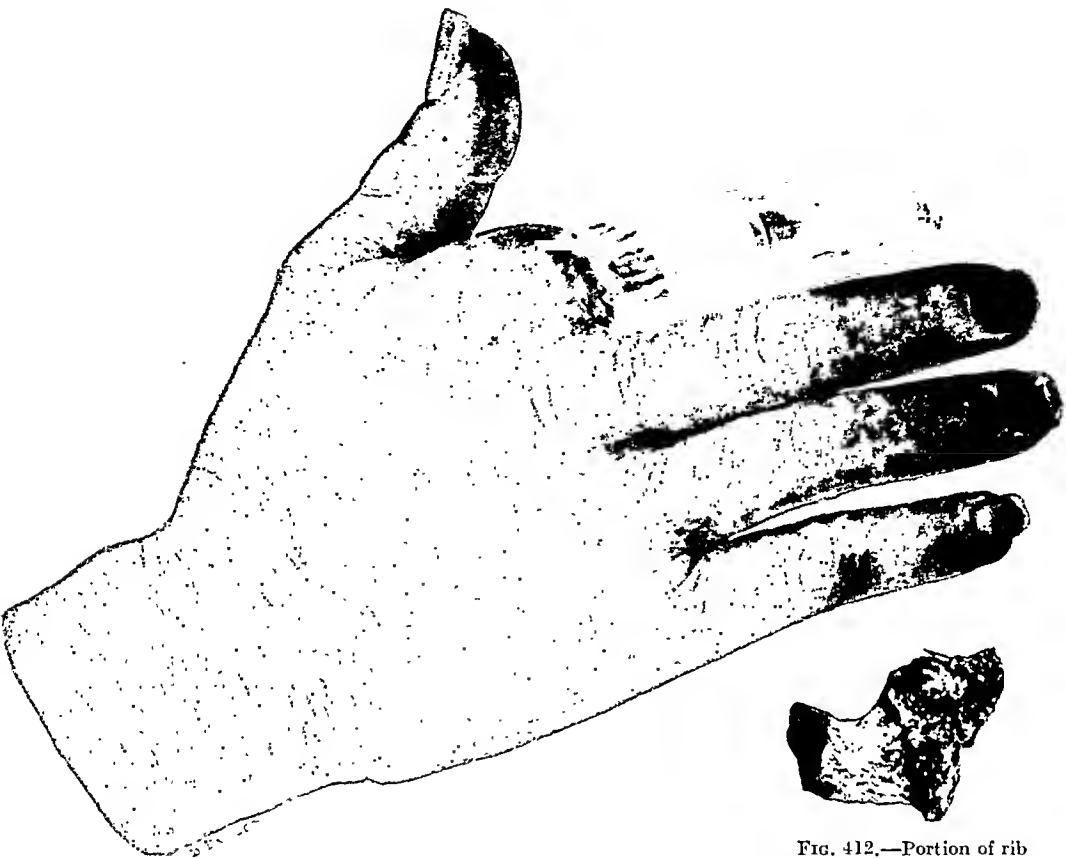


FIG. 411.—*Case 1*. Condition of hand before operation.

FIG. 412.—Portion of rib excised in *Case 1*, showing groove in which the lower trunk of the brachial plexus was found.

AFTER-HISTORY.—The operation was followed, as is usual in these cases, by very prompt relief of pain, and the man in fact slept through the following night without assistance. The hand showed a rapid improvement, and at the end of a fortnight it was evident that all the fingers were recovering with the exception of the index, at the base of which a striking line of demarcation had appeared.

There was steady improvement in the condition of the hand, and at the end of three months a distinct pulse was to be traced in the superior profunda, with obvious enlargement of this vessel. Shortly afterwards the index finger came away at the metacarpo-phalangeal joint, and small portions of the tips of the middle and ring fingers also exfoliated.

The hand is now—eight months after the operation—everywhere of normal colour, and excepting for the loss of the portions mentioned is normal. No pulse other than that of the superior profunda has been felt at any time. The improvement in the man's condition is very apparent: he rapidly recovered in weight the

stone and a half which he had lost in the latter part of his illness, and has now returned to his work.

Two similar cases have been described in detail by one of us (E. D. T.),¹ and a brief summary of these is as follows:—

Case 2.—Male, age 27, admitted to the Manchester Royal Infirmary on March 13, 1912, with the following history: For twelve months he had noticed that his right index finger was numb and white. This condition gradually extended to the thumb and middle finger, and by the end of the year had involved the whole of the hand. No arterial pulse was felt in the main vessels up to a point one inch below the anterior axillary fold. The pulseless arteries could be felt as solid cords. Pulsation in the axillary and subclavian arteries was very marked, and the case had in fact been sent for consultation with a diagnosis of subclavian aneurysm. The cervical rib was removed, and when the man was examined some twelve months afterwards, recovery was complete and he was doing his full work. In this case the inferior profunda was soon felt to be enlarged, and in fifteen months normal pulsation returned in radial and ulnar arteries, but the lower half of the brachial artery remained occluded.

Case 3.—Female, age 29, admitted to the Manchester Royal Infirmary on Sept. 16, 1912, complaining of deadness and loss of use in the right arm. Six months before admission the tip of the index finger became numb and painful, and soon afterwards a large portion of its pulp became gangrenous and sloughed off. Within a month the whole hand was cold and numb. On examination the arterial pulse was not found below the level of the insertion of the coraco-brachialis, but from this point upwards the arteries were normal. A cervical rib of considerable size was removed, and at the end of six months recovery was complete and the patient was back at her work as a weaver. In this case, although colour and use of hand were perfect, no pulse could be felt one year afterwards at any point below the occluded brachial artery. A portion of the radial artery was excised, and the histological findings were described by Todd.²

The similarity of these three cases in their symptoms, physical signs, and recovery after operation is very striking. In each case the patient was right-handed, and the condition is obviously related to the presence of a cervical rib on the side most used at work. It is not easy to understand why the index finger leads the way in the development of the disease; there does not appear to be an anatomical explanation of this fact. The cases agree entirely in the very gradual onset, the initial pallor, coldness, and numbness, the occurrence of gangrene, and the cessation of all pulse at a point about the junction of the axillary and brachial arteries. They all agree, finally, in the very complete recovery which has followed on the removal of a cervical rib.

What, then, is the explanation of this clearly defined symptom-complex? When one turns to consult the text-books on surgery, one is surprised to find that where any explanation is given it is usually that of direct pressure on the subclavian artery. We maintain that any such explanation is untenable: it will not bear critical examination for a moment. It is, in the first place, most unlikely that in a healthy patient the sudden interruption of the subclavian artery by ligature would produce gangrene of even as much as a finger-tip, and it is ridiculous to suggest that any very gradually increasing pressure on the vessel would cause such a result. One of us had occasion recently to ligature the third stage of the subclavian artery together with the transversalis colli and suprascapular arteries in a man of 59 with advanced

atheroma, and not the least threat of gangrene supervened. Further, the most superficial examination of these cases shows that a strong pulse is present in both the subclavian and axillary arteries. This pulse is so marked that the patients have at times been labelled with a diagnosis of "aneurysm of the third stage of the subclavian artery".

In each of the three cases mentioned above the operator has approached the artery with a minimum disturbance of the parts, and in none of the three cases has there been the slightest evidence of any pressure on the vessel, even with the shoulder fully depressed in the classical position for ligature of the artery. It is quite plain that the explanation of pressure on the artery will not do, and we must seek the cause in the relation of the lowest trunk of the plexus to the cervical rib.

In this connection there is another observation which has an important bearing upon the question, and that is the distribution of the vascular occlusion. It is very significant that in each of the three patients this ended abruptly in the region of the lower border of the insertion of the pectoralis major muscle. This means, in the light of recent anatomical research, that the occlusion was limited to those vessels receiving their vasomotor supply from the peripheral nerves, and did not appear in the subclavian and axillary arteries, which are innervated by sympathetic fibres extending out to the arm as a perivascular plexus.

The nerve-fibres accompanying the two large arteries, subclavian and axillary, are remote from the rib and could not be exposed to irritation, or pressure from a rib, and the vessels which they supply were in all three patients quite healthy and normal—whereas the brachial and forearm arteries, which are innervated by sympathetic fibres entering the trunks of the brachial plexus, were occluded. The distribution of the vascular disturbance gives a further reason for studying the lowest trunk in order to explain the symptom-complex which these patients manifest. We are at once faced with the problem of why in one case of cervical rib the symptoms may be purely sensory and motor, whilst in another the results are predominantly thrombotic.

Todd³ was the first to suggest that the cases with vascular symptoms are due to the pressure of the cervical rib upon that portion of the sympathetic which enters the arm via the lowest trunk of the plexus. There can be little doubt that Todd's explanation is the correct one, but it falls short of a complete explanation inasmuch as it does not attempt to explain the mechanism of the thrombosis. Further, there must be some reason why, out of a number of cases suffering from pressure on the lowest trunk of the plexus, only a small proportion will show vascular complications.

With the object of determining the position of the unmyelinated sympathetic fibres in the lowest trunk, transverse histological sections were prepared of this part of the plexus, at the point where it was in most intimate relation with the first rib, on both sides, in eight dissecting-room subjects. It was found that usually the sympathetic fibres were not confined to one particular part of the trunk, but were scattered amongst the myelinated fibres. There was, however, a slight tendency for them to reside principally around the circumference of the trunk. In one case the sympathetic fibres formed a

distinct and separate bundle in the inferior part of the trunk—that is, nearest to the rib (*Fig. 413*). The appearance suggested that the fusion of sympathetic fibres with the lowest trunk was at a more distal point than usual, and that in the region where the section had been cut they had not yet become fully incorporated with the other constituent fibres of the trunk. Todd has reported a similar finding in certain cases.

Such a position would clearly cause the sympathetic fibres to be more exposed to the risk of irritation or pressure by the rib than the motor and sensory fibres would be, and may explain why occasionally in patients suffering from rib pressure the vascular symptoms predominate and may be the only ones.

It appears evident that the arrangement of the sympathetic fibres in the lowest trunk is not absolutely constant, and that they occasionally form a separate bundle in apposition with the rib, where they may be exposed to friction or pressure. In a previous paper¹ we have considered the factors which induce an abnormally intimate relation between the lowest trunk and a cervical or first dorsal rib and thus lead to irritation or compression of the constituent fibres in the trunk. Briefly these may be summarized under three heads: (1) The anatomical relationship of the lowest trunk and rib; (2) Descent

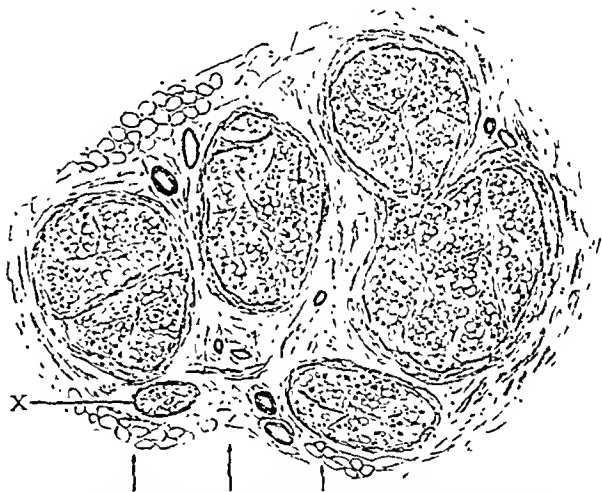


FIG. 413.—Section of lower trunk of brachial plexus in which sympathetic fibres were in an isolated bundle in relation with rib. X = position of bundle: the arrows indicate the region exposed to rib.

of the shoulder during development; (3) Muscular support of the shoulder. Excluding the cases due to trauma, we believe that weakness and loss of tone in the muscles which support the pectoral girdle, especially the trapezius, are the important causes of the onset of the symptoms. In slight degrees of depression of the shoulder the lowest trunk will be only slightly compressed, and friction, with consequent irritation of the fibres, will ensue. When the depression of the shoulder is considerable, progressive compression of the lowest trunk is brought about, with consequent paralysis of vasomotor, motor, and sensory fibres.

The initial symptoms in the three cases recorded were pallor and hypothermia, which indicate vasoconstriction, from irritation—not paralysis—of the sympathetic fibres. It is important to bear in mind that *paralysis* of vasomotor fibres never seems to induce vascular changes. One of us

(J. S. B. S.) followed over many years a number of irreparable divisions of peripheral nerves, and frequent examinations of the patients failed to reveal any tendency to arterial occlusion in consequence of paralysis of the vasoconstrictors. On the other hand, partial division and irritation of peripheral nerves has been found on several occasions to be succeeded by vascular changes, and these have been shown histologically.⁵ Previous observers seem to have assumed that in the cases under consideration the sympathetic fibres were paralysed by the pressure of the rib, but there are at least three facts which oppose such a contention: (1) The initial pallor and symptoms of vasoconstriction; (2) The knowledge that paralysis of sympathetic fibres, even when long continued, never leads in itself to vascular occlusion, whereas irritation of a peripheral nerve has been shown to be associated with arterial changes; (3) The immediate relief of the vascular symptoms after operation—which could not occur if the sympathetic fibres had been paralysed for so long a time, since degeneration would have been inevitable.

There is good reason, therefore, to believe that irritation of the vasoconstrictor fibres in the lowest trunk of the plexus by a rib is the fundamental cause of the pallor, hypothermia, and vascular pain. There arises the question whether the irritation and consequent prolonged vasoconstriction can induce arterial occlusion. We would put forward the suggestion that the spasm of the arteries must inevitably induce constriction or even obliteration of their vasa vasorum. Prolonged narrowing or occlusion of the vasa vasorum is likely to cause nutritional changes in the walls of the arteries and to lead ultimately to thrombosis.

The matter might be put to experimental test, but the difficulty is to find any method by which long-continued irritation of sympathetic fibres can be artificially produced. It is hoped in the near future to devise some method by which this may be accomplished. In the meantime perhaps the nearest approach which can be made to an experimentally produced constant spasm of the arterial wall is by using ergot. Dr. Maedonald very kindly undertook for us the treatment of two white Leghorn cockerels by ergot in the Department of Pharmacology in the University of Manchester. In the one case the bird was given $\frac{1}{2}$ grm. of ground ergot in its food daily for two months; the second bird was given a daily intramuscular injection of 0.2 c.c. of a 0.1 per cent solution of ergotamine tartrate (Stoll) during a period of six weeks. The big florid combs shrank steadily until they were reduced to one-tenth of their original size and their appearance was that of dirty chamois leather. At this stage sections of the combs were cut, and these show that the arteries are the seat of a proliferative endarteritis, whilst the lumen is occupied by a hyaline thrombus. These appearances are shown in *Fig. 414*. It is noteworthy that the change is most marked, and evidently begins, in the smaller arteries, extending subsequently to the large axial artery of the comb. The veins are quite unaffected. Our findings in this respect agree entirely with those given by von Recklinghausen.⁶

It is likely, therefore, that the vascular changes when seen in cases of cervical rib are due to prolonged spasm and resemble those of ergotism. Clinical observation of the cervical rib cases shows that the changes begin, and are always most extreme, in the small vessels at the periphery. The

VASCULAR COMPLICATIONS OF CERVICAL RIB 563

thrombosis here induced spreads subsequently to the larger vessels. In one patient this process was evident. When first seen he had a good pulse in the brachial artery at the bend of the elbow, but during the few days he was awaiting admission to hospital the thrombosis gradually ascended and involved the whole of the brachial artery.

Our interpretation of these cases of gangrene associated with the presence of a cervical rib is that in some patients the bundle of sympathetic fibres which is destined to innervate the peripheral vessels of the arm lies in the lowest trunk of the plexus in such a position that it is exposed to pressure from the rib. This pressure causes irritation of these fibres, with consequent spasm of the arterial wall. The persistent spasm of the wall of the artery must embarrass the vasa vasorum and lead to changes in the health of the

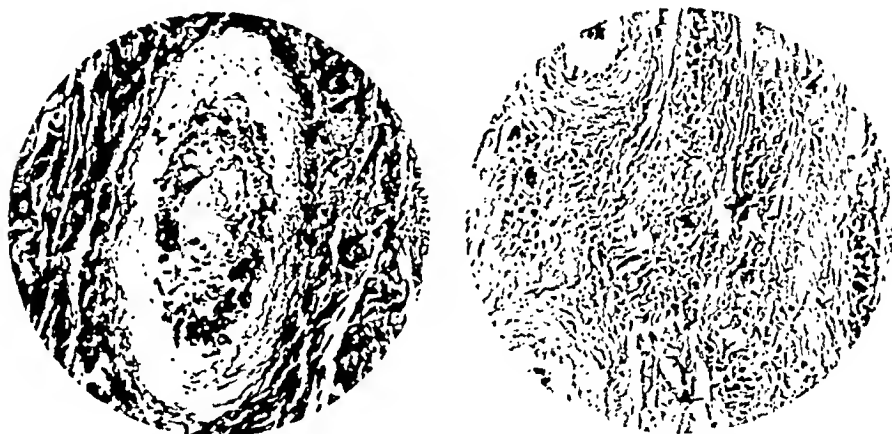


FIG. 414.—Sections of the combs of Leghorn cockerels after injection of ergotamine tartrate, to show proliferative endarteritis and, in that on the left, the presence of a hyaline thrombus in the lumen. ($\times 200$.)

arterial wall. The thrombosis is secondary to these changes. That portion of the main trunk (subclavian and axillary) which lies above the lower border of the pectoralis major tendon is innervated from a different source which has no possible relation to a cervical rib, and therefore this section of the main trunk remains always open and healthy in these cases.

If this hypothesis is tenable, it is possible to advance reasons for the small proportion of patients who manifest vascular complications, since all the following conditions are essential for the development of the complications: (1) Unusually intimate relationship of sympathetic fibres and rib; (2) Such depression of the shoulder as will lead to irritation—and not paralysis—of the sympathetic fibres in the lowest trunk; (3) The irritation must be of considerable duration.

It has long been known that the sensory and motor symptoms associated with cervical rib can be produced by a normal first rib, and we have published a series of these cases.⁴ So far no case with vascular symptoms appears

to have been traced to a normal first rib, but there is no reason why this should not happen.

Of many cases of cervical rib with symptoms, few will show vascular disturbance, since few have the peculiar distribution of the sympathetic fibres which is essential to the development of vascular symptoms. Hence, in those much rarer cases in which the pressure is due to a normal first rib, the chances of unusual sympathetic distribution coinciding with the condition are of necessity remote. It is not unlikely that such a case may be reported in the future, and in the meantime it is worth while to remember that if a case presents vascular symptoms with no X-ray evidence of cervical rib, a normal first rib may none the less be the cause.

REFERENCES.

- ¹ TELFORD, E. D., "Cervical Rib with Vascular Symptoms", *Lancet*, 1913, ii, 1116.
- ² TODD, T. W., *Jour. Nerv. and Ment. Dis.*, 1913, xl, 440.
- ³ TODD, T. W., *Lancet*, 1913, i, 1371.
- ⁴ STOPFORD, J. S. B., and TELFORD, E. D., *Brit. Jour. Surg.*, 1919, vii, 168.
- ⁵ STOPFORD, J. S. B., *Lancet*, 1918, March 30.
- ⁶ VON RECKLINGHAUSEN, F., *Handbuch der allgemeinen Pathologie des Kreislaufs und der Ernährung*, 1883, 349, Stuttgart.

OSTEOMA OF THE FRONTAL SINUS:
WITH PARTICULAR REFERENCE TO ITS INTRACRANIAL
COMPLICATIONS, AND WITH THE REPORT OF A CASE.*

BY GEORGE ARMITAGE, LEEDS,
 ROCKEFELLER FELLOW, 1929-30.

OSTEOMATA of the frontal sinus present many interesting problems, mainly on account of the effects which they produce by pressure upon neighbouring structures. Of primary interest to the rhinologist, they may extend by their characteristically slow and progressive growth into the domain of the ophthalmologist or the surgeon. In olden times, if small, they often passed unnoticed during life, only to be accidentally discovered by anatomists; if larger, they either became outwardly visible or produced symptoms and signs referable to the frontal sinus and nose, the eye, or the brain. The use of X rays has been of great help in establishing their presence, determining their extent, and appreciating the possible complications to which they may give rise. It is the importance of the intracranial complications which calls for emphasis.

A review of the literature concerning these interesting tumours gives much information as to their form and extent. This report will deal primarily with a large osteoma of the frontal sinus complicated by a ventricular pneumatocele and cerebrospinal-fluid rhinorrhœa. It will be recalled that in 1927 Dr. Cushing¹ gave a detailed report of four cases of orbito-ethmoidal osteomata having intracranial complications, and discussed in particular the treatment of these cases.

HISTORICAL INTRODUCTION.

Osteomata of the orbit have, according to Parsons,² been recorded since 1506, when Veiga first called attention to them, though the difference between primary bony tumours of the orbital cavity and those invading it secondarily could not then have been appreciated. The occurrence of bony tumours in connection with the frontal sinus has long been recognized both in domestic animals, in which they are comparatively common, and in man. Usually of small size, they occasionally attain truly alarming dimensions. This is small wonder if the osteoma became so large as to occupy one half of one hemisphere and to project considerably over the forehead, nose, and eye, as occurs in a specimen reported by Paget³ which is now in the Museum at Cambridge. Yet another enormous tumour, occurring in an ox—the specimen of which is in the Museum of the Royal College of Surgeons of England (*Path. Spec.* No. 3216)—is described by Paget as “a great spheroidal mass of ivory

* From the Surgical Clinic of the Peter Bent Brigham Hospital, Boston, U.S.A.

measuring $8\frac{1}{2}$ inches in diameter and weighing upwards of 16 pounds". In veterinary literature some of these cases have been reported as 'ossified brains'.

As far back as 1799, Home⁴ reported examples of tumours of the frontal sinus, and there are to be found illuminating pathological descriptions of frontal osteomata in the subsequent writings of Mackenzie,⁵ Bouyer,⁶ Rokitsansky,⁷ Weiss,⁸ Cruveilhier,⁹ Virchow,¹⁰ and Hilton.¹¹ Efforts to remove these tumours were occasionally made, particularly by ophthalmic surgeons, but the operations were usually left incomplete and were attended with a high mortality. The difficulties appear to have been threefold: (1) The compact, hard, ivory nature of the tumours rendered them extremely resistant to any form of mechanical interference by saw or hammer and chisel; (2) The inability to determine by clinical examination alone the full extent of the mass; and (3) The extreme liability to a post-operative intracranial infection.

Paget³ in 1851 reported a case occurring in a girl of 20 who suffered from exophthalmos due to an osseous growth in the upper and inner part of the orbit. It had been gradually increasing for three years, producing severe pain in the eye and side of the face. An attempted radical removal failed, and it was with great difficulty that an anterior portion was sawn off. The patient died shortly afterwards from generalized suppurative meningitis.

Roux¹² abandoned an attempt to remove a similar tumour in a young man, with the customary fatality from meningitis. The tumour was removed at autopsy by Nélaton and is now in the Val de Grâce Museum in Paris. Weiss⁸ reported this case to the Société Anatomique in 1851, and Dolbeau¹³ also describes and depicts it in his admirable memoirs on the exostoses of the frontal sinus written in 1871.

An unsuccessful attempt at removal by hammer and chisel of what was clinically a small tumour in a lad of 14 was made by Knapp¹⁴ in 1861, with eventual death from meningitis in seven weeks. At autopsy this tumour was found to be much more extensive than was expected, measuring 59 by 55 by 54 mm., filling the left frontal sinus completely, the right partially, part of the ethmoidal cells, and the whole of the left and part of the right anterior cranial fossa.

There is an excellent example of a large osteoma of the frontal sinus to be found in the Warren Museum of the Harvard Medical School (*Path. Spec.* No. 1464) (*Figs.* 415-417). This occurred in a healthy young farmer who entered the Massachusetts General Hospital in 1864. There was a history of a severe blow on the forehead sustained while skating three years previously. The tumour measures 12.5 by 12 by 12.5 cm. Attempted removal, in the course of which four chisels were used with failure to penetrate the growth, was abandoned. Death in coma took place five weeks later, and at autopsy an abscess was found in the frontal lobe.

An attempt made by Jobert to remove, with a trephine, an 'exostosis' of the frontal sinus was abandoned on account of the extreme hardness of the growth. The patient died, and Dolbeau¹³ refers to the specimen, which is in the Musée Dupuytren in Paris.

Arnold¹⁵ in 1873 reported two cases, in one of which death occurred without operation. Simon operated on the other case, with the outcome of

post-operative meningitis and death, an abscess having formed in the left frontal lobe with rupture into the lateral ventricle. The dimensions of this

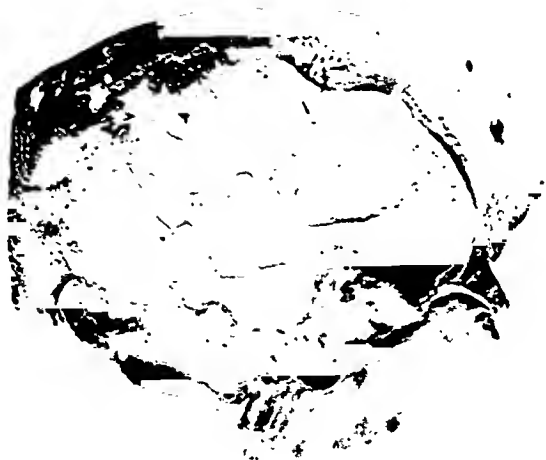


FIG. 415.—Large frontal-sinus osteoma, antero-posterior view. The flat surface in the centre was the result of attempted removal at operation. ($\times \frac{3}{4}$.) (*Path. Spec.* No. 1464. *Warren Museum, Harvard Medical School.*)



FIG. 416.—The same, postero-anterior view. ($\times \frac{3}{4}$.)

tumour were 9 cm. vertically, 7.5 cm. laterally, and 7 cm. antero-posteriorly, bearing a marked resemblance to the measurements of the tumour in the case to be subsequently described. Banga¹⁶ also mentions a case operated

upon by Socin in 1871, with death from meningitis on the tenth day after operation.

Although two cases of spontaneous recovery following infection and suppuration with extrusion of the tumour are reported by Hilton¹¹ and Lucas,¹⁷ the credit for the first successful operation, in 1867, belongs to Dolbeau,¹³ whilst a second, with an equally good result, was carried out by Knapp¹⁸ in 1880.



FIG. 417.—The same, lateral view, showing the limits of the anterior and posterior extensions. ($\times \frac{3}{4}$.)

Up to and including 1880, surgery, as judged from reported cases in the literature had been attended by two cures, and in addition two cases had spontaneously cured themselves by sloughing—one of them after operative interference. This state of affairs undoubtedly influenced Berlin¹⁹ when he wrote, in 1880, that enucleation of the eye should be resorted to when an osteoma of the frontal sinus has broken into the orbit, rather than any attempt to remove the tumour. Knapp,¹⁸ in 1881, however, strongly advocated surgical removal, and stated that osteomata of the frontal sinus may and ought to be removed by subperiosteal enucleation, with careful avoidance of injury to the dura mater, as long as they are moderate in size.

However, towards the close of the last century, owing to the general adoption of Listerian principles, the prognosis following removal of osteomata of the frontal sinus underwent a radical change. An excellent account of the history of osteomata of the peri-orbital pneumatic cavities, with a report of the successful removal of two osteomata of the orbit, one originating in the frontal and the other in the ethmoidal air-cells, was written by Andrews²⁰ in 1887. In 1898 Tauber²¹ reviewed the literature and found that 20 cases of frontal-sinus osteomata had been removed by operation, with 9 deaths—a mortality of 45 per cent. There is always present, then, a liability to infection, and, as in the prognosis of compound fractures of the skull, the important factor is whether the dura mater is intact or not—a point to which Knapp¹⁸ drew attention. If, however, as in the example reported below, there is a free communication from the outside air, not only through the dura mater, but in addition through the cerebral substance into the ventricle, the case assumes far more serious proportions. In these circumstances the lesion can only be attacked with safety from above by way of the cranial chamber.

ETIOLOGY.

Frontal osteomata are more common in males than in females. This may be due to the fact that the sinus is on the average considerably larger

in the male, and therefore the seat of greater developmental activity; also it is more liable to external trauma. Fifty per cent of cases are said to occur during adolescence, and 80 per cent before the age of fifty years; that is, these tumours occur in young people, thereby resembling the occurrence of bony tumours elsewhere in the body. The second and third decades cover the period of life in which they are most likely to occur. These tumours of the frontal sinus are rare, but their relative frequency is difficult of estimation. Certainly before the advent of almost routine X-ray examination in cases of sinus trouble, several small 'latent' osteomata were missed. In 1881 Bornhaupt²² reviewed the literature and came to the conclusion that the condition was more frequent than was formerly supposed. Knapp²³ in 1880 reported only 4 cases with intra-orbital extension in 56,000 patients; and in 1881¹⁸ he reported 11 cases, his own single case, with cure following operation, being the first reported in America. Andrews²⁰ in 1887 found only 8 examples of bony growths of the orbit in nearly 430,000 ophthalmological patients. In 1905 Hucklenbroich²⁴ collected 54 cases of true frontal-sinus osteomata; in 1907 Gerber²⁵ reported 87; in 1918 Sewall²⁶ 93; in 1925 Lee²⁷ 100; in 1928 Harris²⁸ 117; in 1929 Browder²⁹ reported one case, to which may be added the case to be reported subsequently, bringing the number to 119. Kikuzi,³⁰ analysing 54 cases of accessory-sinus osteomata, found that 26 were of the frontal sinus; 11 ethmoidal; 10 antral; 5 of the sphenoid recess; and 2 of the nasal cavity. On the other hand, Gerber²⁵ gives the proportion of those arising in the ethmoid to those arising in the frontal sinus as 12 : 8. In 1918 Culbert³¹ collected 215 cases of accessory-sinus osteomata reported since 1748, and stated that nearly half of them were of the frontal sinus. It is probable that frontal-sinus osteomata represent about 35 to 40 per cent of all accessory-sinus osteomata. The reason for this high figure, together with the causation of growth of these tumours, has been for a long time a vexed question and still remains unsettled. Many ingenious theories have been advanced in explanation, the most notable being:—

1. Embryological or Developmental.—It will be recalled that the frontal bone is ossified in membrane, whereas the ethmoid bone is ossified in cartilage. The frontal bone is the centre of marked developmental activity until well after puberty. Whilst traces only of the frontal sinuses are met with in the second year, they can be definitely distinguished at the seventh year, but do not attain complete development until about the sixteenth year. This state of affairs, coupled with the close proximity to the ethmoid bone, appears to favour the liability to occurrence of cartilaginous 'rests' in accordance with Cohnheim's original theory of the origin of new growths. In 1873 Arnold¹⁵ advanced this cartilaginous 'rest' theory, pointing out, as did Cohnheim, that where different tissues come together there is a predisposition towards tumour growth. There is, however, much to be said against this theory. No evidence of cartilage is found in connection with these tumours, and they do not always arise from the ethmoidal bone. It was Rokitansky⁷ who first supposed that these tumours had their origin in enchondromata. First, he thought the endosteum of the frontal sinus produced cartilage, forming enchondromata, which later became ossified, forming osteomata. Bornhaupt²² in 1881 came to the conclusion that these tumours were of periosteal origin, although unable

to settle the question of their etiology. Sappey (quoted in Dolbeau's article¹³) lent support to the periosteal idea of their origin, stating that the periosteum in this region is peculiarly liable to ossification.

2. Traumatic.—Though a history of trauma, recent or remote, is often obtained, its direct relation to the osteoma is impossible of proof. Many writers, nevertheless, including Dubar,³² de Taranto,³³ and Miodowski,³⁴ favour external trauma as an important etiological factor.

3. Infective.—Axenfeld³⁵ in 1904 noted the frequency with which catarrh of the mucous membrane of the sinus complicates the presence of these tumours. Hucklenbroich²⁴ stated that 37·8 per cent were definitely associated with sinusitis. Gerber²⁵ in 1907 expressed the opinion that they were the result of repeated infection, transient but more or less frequent; and Wray³⁶ in 1916 contended that sinusitis was the exciting cause in all osteomata of the accessory sinuses. He presented a specimen showing a bony excrescence which had not reached the stage of osteoma.

Syphilitic infection has been considered by some to be the etiological factor. Prior to the advent of the Wassermann test, syphilis was often quoted as the cause, but since then one rarely finds it given. Two cases reported by Culbert³¹ and Mackenty³⁷ appear to be the only ones in which a positive Wassermann test was found.

As a matter of fact, it is not precisely known what the etiological factors actually are. So far as concerns infection, it is probable that the chronic irritation of the mucous membrane is a sequel of the lesion rather than its cause. What is more, infection rarely accompanies the osteomata of the accessory sinuses in their earlier stages.

PATHOLOGY.

Osteomata of the frontal sinus, as is almost invariably the case with bony tumours growing in connection with the cranial bones, are of the hard, ivory, compact variety—so-called 'exostosis eburnea'. They are connected to the bony wall of the sinus usually by a small pedicle, but occasionally they appear to have no point of attachment. Microscopically they show a concentric or irregular arrangement of the bone corpuscles, with a scantiness or absence of Haversian canals. Some specimens show a cancellous interior with a hard bony shell around it.

In 1856 Cruveilhier⁹ gave the first description of any real value of these tumours under the title of 'corps osseux enkystés', believing that they originated in the diploë between the two tables of the skull, either forcing the inner and outer tables before them as a capsule or rupturing through them. In 1865 Virchow,¹⁰ while giving credit to Cruveilhier for the idea that they arise in the diploë of the skull bones, took issue with the term 'corps osseux enkystés' and preferred not to separate them from the 'enostoses', a term which he introduced in contradistinction to the exostoses.

In 1907 Gerber²⁵ pointed out that these tumours present certain similarities with nasopharyngeal polypi, in that they are histologically benign but locally malignant—a feature referred to by Paget when describing the horrible complications to which they may give rise. This is further exemplified

by Bland-Sutton's³⁸ case, in which the following train of events occurred: (1) A swelling the size of a pea noticed at birth; (2) At 9 years the eye was totally destroyed; (3) At 25 the skin of the brow sloughed over; (4) At 35 the tumour fell out of the orbit, with spontaneous cure. Occasionally, also, these tumours show a liability to local recurrence after apparently complete removal. Thomas³⁹ reports such a case in a boy of 14, in which following an injury there was unusually rapid growth, so much so that within three months of the injury a dense osteoma had occurred involving all the left frontal sinus, extending into the orbit, and upward above the sinus and beyond the mid-line somewhat over the right orbit and into the right ethmoidal region. This was removed completely, and within nine months it had recurred. X-ray examination showed it to be even larger than previously. Three years after operation, radiograms showed that the tumour had grown back so extensively into the brain cavity as to be inoperable.

Barnhill⁴⁰ describes a case in a girl of 16 with an osteoma of the right frontal sinus of one year's standing which, as found at operation, grew from the region of the fronto-nasal duct. Ingersoll,⁴¹ commenting on this case, points out the known tendency of these osteomata growing from the region of the fronto-nasal duct to recur, and as a result expresses a hesitancy to operate in such cases. Possibly when these tumours in young people show rapid growth and are attached to this region they are frankly sarcomatous.

Two additional points regarding the pathology to which scant, if any, attention has been called have emerged from the case reported below: (1) The presence of mucous membrane between tumour and normal bone serves to establish the line of cleavage between them: and (2) The mucous secretion shows a tendency to collect into cyst-like cavities called mucocoeles which project through the dura mater into the brain substance.

CLINICAL ASPECTS.

Many osteomata of the frontal sinus remain small and pass undetected. It is characteristic of these tumours that they become steadily larger, so that sooner or later the majority produce subjective or objective evidence of their presence. They enlarge in a centrifugal manner, gradually destroying by pressure atrophy the limiting bony walls of the sinus in which they are contained. Whilst still confined within the greatly expanded sinus they manifest themselves by symptoms and signs resulting from inflammation and obstruction. In their onward march beyond the limits of the sinus they may extend anteriorly on to the forehead, across the mid-line through the medial septum into the opposite frontal sinus, or into the nasal, orbital, zygomatic, temporal, or cranial fossæ. Whichever direction they may take, they produce evidence of their invasion by pressure effects, causing obstruction, displacement, or destruction, coupled with the formation of mucocoeles. For example, a frequent extension of these tumours is into the orbital cavity. By pressure they displace the eye downwards, forwards, and outwards, and interfere with the normal ocular movements, with consequent diplopia. Infection of the orbital cellular tissues is not uncommon. As a result of extrusion of the eye, the normal protective movements of the eyelids are impeded, with resulting

corneal ulceration and its sequelæ. But it is the effects of direct extension backwards into the cranial cavity that are of particular interest. It has long been known that these tumours may produce frontal-lobe symptoms by pressure thereon—namely, headache, dispositional changes, irritability, depression, mental dullness and apathy, together with the formation of large intracranial mucocœles. The case to be reported goes one stage farther, with rupture of a mucocœle into the lateral ventricle and with subsequent pneumatocele.

CASE REPORT.

Osteoma of the frontal sinus with intracranial complications. Intracranial mucocœle communicating with the ventricle, causing cerebrospinal rhinorrhœa, and radiographically demonstrable filling of the ventricular system with air. Removal of tumour in two sessions. Repair of cerebrospinal fistulæ at a third. Recovery.

T. A. T. (Surgical No. 35928), male, age 36, was admitted to the Peter Bent Brigham Hospital on March 5, 1930, on the advice of Dr. Hanrahan, of Rutland, Vermont, complaining of periodic 'filling up' at the root of the nose causing dull headache, followed and relieved by a watery discharge from the nose.

HISTORY.—The patient was a healthy, vigorous man, an electrical supervisor by occupation, with no history of any preceding illness which might conceivably



FIGS. 418, 419.—The patient on admission to hospital.

have had any bearing upon the present condition. There was no history of difficulty or injury at birth, of trauma, or syphilitic infection. He had been married for fifteen years, there being five children, all living and well, whose ages ranged from 14 to 6 years.

He had consulted Dr. Cushing in 1913 on the recommendation of Dr. W. P. Clough, of Sutton, New Hampshire, because of a 'hard projecting lump' on his forehead which had first been noticed by his mother six years previously and which was slowly increasing in size. After examination, including an X-ray study, a

diagnosis of osteoma of the frontal sinus was made. An operation was advised but declined.

In the seventeen years' interval the tumour did not undergo any marked outward change. There had been a predisposition to attacks of 'grippe' at intervals during the winter months which caused him undue trouble, leaving him very weak and easily fatigued afterwards. During the last twelve months a subjective sensation of periodic 'filling up' behind the root of the nose associated with a dull headache had occurred, to be followed by the discharge of a watery fluid drop by drop from the left nostril only, with relief of both the discomfort and headache. This post-nasal discomfort had occasioned extreme irritability of the nose, causing the patient to get into the habit of blowing his nose violently and frequently in an attempt to rid himself of his discomfort. The headache was generalized, but maximal bitemporally. The patient had also experienced, when the 'filling up process was incomplete', as he thought—probably correctly—a subjective sensation of splashing within the head. The nasal discharge was watery, clear, colourless, odourless, and tasteless.

In January, 1930, he had a particularly severe attack of 'grippe' in which quite suddenly he became semiconscious, extremely drowsy, and stuporous. His memory of this attack is entirely lacking, but his wife states that he remained in this condition for about thirty-six hours, gradually recovering normality except for noticeable increase in irritability. It was this attack that led to his re-entry into the hospital.

ON EXAMINATION.—Physical examination revealed the presence of a stony-hard bony swelling on the forehead, rather more to the left than to the right of the mid-line, between the root of the nose and the hair line (*Figs. 418, 419*). It was not attached to the skin, which was normal and freely mobile over it, but it was fixed deeply to bone. Roughly circular in outline, it measured approximately 6 cm. in diameter, with a maximum elevation of 2 cm., gradually shelving off to the general level of the forehead at its margin. The surface appeared uniformly hard and slightly uneven. Nasal examination was negative. The fundi and visual fields were normal.

X-ray Examination (*Figs. 420, 421*).—This was made by Dr. Sosman, who reported as follows: "Right stereo antero-posterior and postero-anterior films of the skull show a thin vault with an unusually dense cauliflower mass filling the frontal sinuses and apparently breaking through both anteriorly and posteriorly. This has the typical appearance of an osteoma, probably arising in the frontal sinus. It measures 6.5 cm. vertically, 7 cm. laterally, and 6 cm. antero-posteriorly. There are slight signs of increased intracranial pressure, and also several collections of air in the left ventricle, the entire left temporal horn being well filled, indicating a communication between it and the frontal sinus. The sella turcica is normal and the mastoids are clear."

DIAGNOSIS.—A diagnosis was made of an osteoma of the frontal sinus complicated by an intracranial mucocoele communicating with the ventricular system, and an operation was recommended.

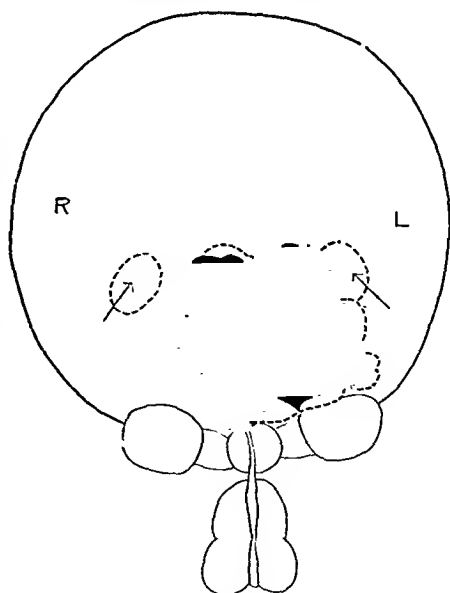


FIG. 420.—Diagram of postero-anterior radiogram on admission, showing: (1) The cumulus-like outline of the tumour mass, bounded in areas by a rim of air in the tumour-expanded sinus. (2) The air-containing occipital horns of the ventricles (marked by arrows).

FIRST OPERATION (March 16).—Novocain-adrenalin infiltration anaesthesia. Left transfrontal approach. Exposure of a large eburnated osteoma. Gradual



FIG. 421.—Left lateral skiagram on admission, showing: (1) The shadow of the tumour in profile with evident extension through the anterior wall (marked by arrow) of the sinus; (2) The air accumulated in the temporo-occipital horn of the right ventricle (T.O.H.).

excavation of osteoma. Piecemeal dislodgement of main portion of the extremely nodular tumour from greatly expanded frontal sinus. Accidental opening of the ethmoidal cells. Disclosure of a large multicystic mucocoele, of which three or four cysts came away with the chief mass of the tumour, completely denuding collapsed frontal lobe and exposing lateral ventricle. Closure with drainage.

Dr. Cushing had hoped to be able to turn down a bone-flap from over the tumour in order to spare the patient, so far as possible, from any subsequent deformity. Accordingly, the usual left transfrontal flap was outlined and the first burr hole was made in the region of the glabella. Immediately bony tumour was encountered, confirming the X-ray evidence that the anterior wall of the frontal sinus was in great part deficient over the tumour, having been destroyed by pressure atrophy. A succession of burr holes were then made into the tumour, and by degrees many nodules of the growth were freed and rocked out of their bed. When in this



FIG. 422.—Showing mucous-membrane-lined fragments of the osteoma removed at the first operation (March 17, 1930). Note mucocoeles (M) in upper part of field.

process the posterior limit of the tumour was finally reached, no trace of the expanded posterior bony wall of the sinus or of the dura mater was visible. Instead, there was a large multilocular cystic mucocele, of which three or four cysts came away attached to nodules of the tumour, with some leakage of purulent-looking contents. In view of this leakage it was considered advisable to withhold further interference with remaining portions of the tumour or with the large mucocele which appeared to extend into the open lateral ventricle. The wound was closed, with gutta-percha tissue drainage at each angle of the incision.

The fragments of the osteoma are shown in *Fig. 422* just as they were removed. Hanging from three or four nodules can be seen the remains of mucous membrane which had formed part of the huge multicystic mucocele which extended so far into the brain as to project directly into the ventricle.

Bacteriological examination of the contents of the mucocele showed them to be sterile.

Post-operative Course.—Cerebrospinal rhinorrhœa from the left nostril commenced almost immediately after operation, and persisted. Daily tappings through the centre of the flap and bony defect were carried out, drawing off approximately 40 to 50 c.c. of blood-stained fluid per diem, with disappearance of all tension of the flap. The drains, being of little value, were removed on the second day. A rise of temperature to 101° rectally with an associated white-cell count of 17,500 occurred within twelve hours of the operation. This fever, after rising as high as 102.8° rectally on the third day after operation, gradually subsided, as did the leucocytosis, so that on the eighth day the chart was normal in all respects. At this time (March 24) X-ray examination was repeated, showing that about two-thirds of the osteoma had been removed, there still remaining a good-sized fragment in the right frontal sinus (*Figs. 423, 424*). In addition there was a loose fragment in the cranial cavity and also very considerable amounts of air in the ventricles.

Valuable information had been obtained as a result of this first operation. Although in the main the tumour was of bony hardness throughout, certain areas were not quite so hard, and yielded more easily when attempts to remove them were made. In addition, the demarcation lines between normal bone and tumour noted in the radiograms were clearly understood, being marked by the presence of mucous membrane. On the ninth day after operation it was therefore decided to attempt removal of the remainder of the tumour.

SECOND OPERATION (March 26).—Reflection of old flap. Piecemeal removal of remainder of tumour. Occlusion of ethmoidal communication by muscle implant. Attempt to remove posterior wall of mucocele projecting deeply into the frontal lobe and ventricle abandoned. Closure with drainage.

It was possible, fortunately, to preserve the paper-thin supra-orbital bony ridge, thereby reducing the inevitable subsequent deformity. The remaining fragments of the tumour (*Fig. 425*), attacked by burrs, rongeurs, and periosteal elevators, were finally rocked out of their bed. The region of the blow-hole was located by a device which would not have been possible had the patient been under inhalation narcosis; the bottom of the huge cavity was covered with a layer of saline solution and the patient asked to hold his nose and to exhale forcibly. Air bubbled up

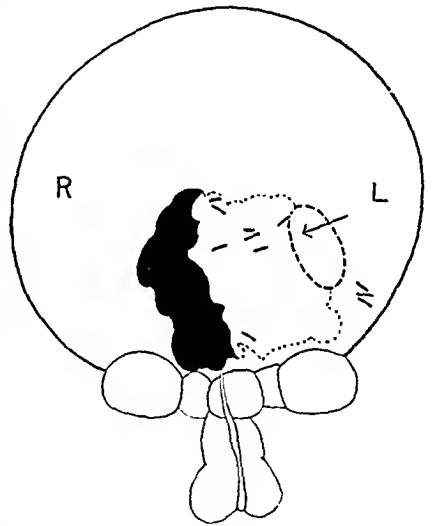


FIG. 423.—Diagram of postero-anterior radiogram (March 24, 1930), seven days after the first operation, showing: (1) The residual portion of the tumour to the right of the mid-line; (2) The air in the occipital horn of the left ventricle (marked by arrow).

through the fluid, making it easy to locate the point of communication into the nose. A flap of mucous membrane was turned back and a muscle stamp implanted under its edges.

Post-operative Course.—This was in the main similar to that following the first operation, except that it did not occasion so much apprehension in view of the

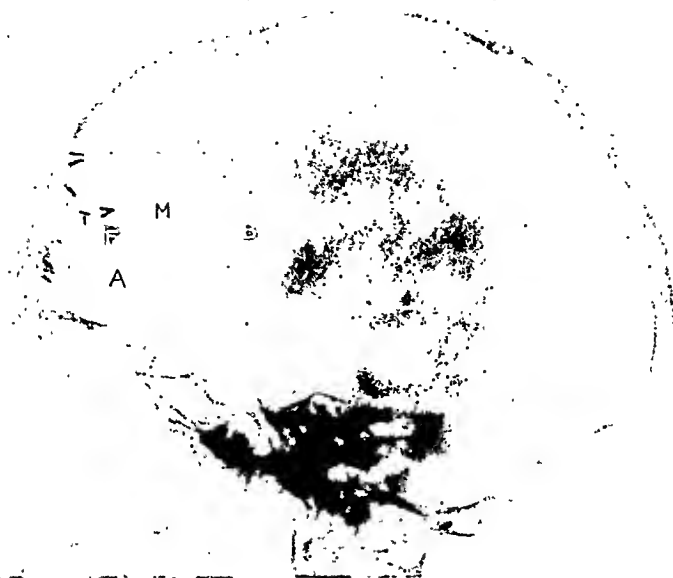


FIG. 424.—Right lateral skiagraph (March 24, 1930), showing the residual tumour with free fragment (F), and with air at A in free space of former bed of tumour, and at B in anterior horn of left ventricle, into which mucocoele (M) projected.

experience already obtained. After removal of the drain on the third day daily tappings were carried out, approximately 50 c.c. of much clearer fluid than formerly being removed. X-ray examination carried out on the sixth day (April 1) showed that the osteoma had been completely removed (Figs. 426, 427). There was no air in the ventricle.

On the ninth day the patient was perfectly well. He was up in a wheel-chair, temperature, pulse, respiration, and white-cell count being normal, and the wound completely healed, with no bulging of the flap. On the following day, however, cerebrospinal rhinorrhoea recommenced, in no way due to any misbehaviour on the patient's part. He had most strictly adhered to instructions to avoid violent movements of the head, blowing his nose, or sneezing. Most disappointingly, however, the discharge continued and increased in amount. On April 8, X-ray examination showed an abundance of air in the ventricle, one irregular collection being situated just beneath the operative area, probably in the mucocoele, whilst another regular collection, estimated at about 15 to 20 c.c., clearly outlined the ventricular system.

In the hope that the discharge of

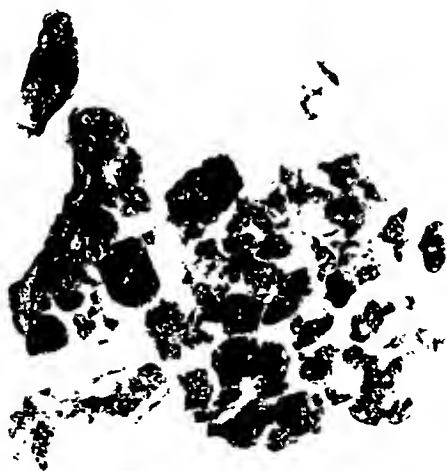


FIG. 425.—Fragments of the osteoma removed at the second operation (March 26, 1930).

cerebrospinal fluid would cease spontaneously. further operative interference was postponed. but no improvement occurred. Further X-ray study carried out on April 22 (Fig. 428) showed exactly the same amount of air in the ventricles as was seen on April 8. and further operative interference was decided upon, because the patient still had the condition for which he was operated upon originally—namely. cerebrospinal rhinorrhœa—although the frontal osteoma itself had been removed.

THIRD OPERATION (April 28).—Exploration disclosed a huge, perfectly dry mucous-membrane-lined cavity with two openings, one into the right nasal cavity and the other into the left. Implants of fascia taken from patient's leg placed over both openings under mucous membrane flap.

The cavity, completely lined with apparently normal-looking mucous membrane, was of exactly the same size as seen at the former operation. In the base of the cavity leading apparently down into the nares, where the previous muscle implant had been made, there was a hole which admitted the tip of the little finger. The muscle implant probably had shrunk. A few drops of fluid irrigated through this hole ran directly into the nasopharynx and were swallowed. The mucous membrane was reflected from around this opening, a fascial stamp about 2 cm. in diameter was used to cover up the hole, and the edges of the mucous membrane were carefully placed

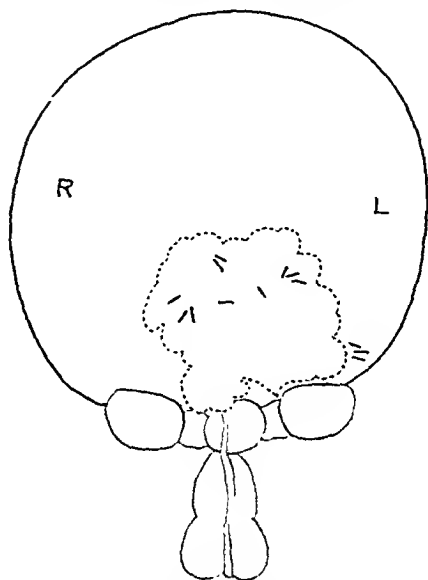


FIG. 426.—Diagram of postero-anterior radiogram, six days after second operation, showing complete removal of tumour. No air in ventricles.

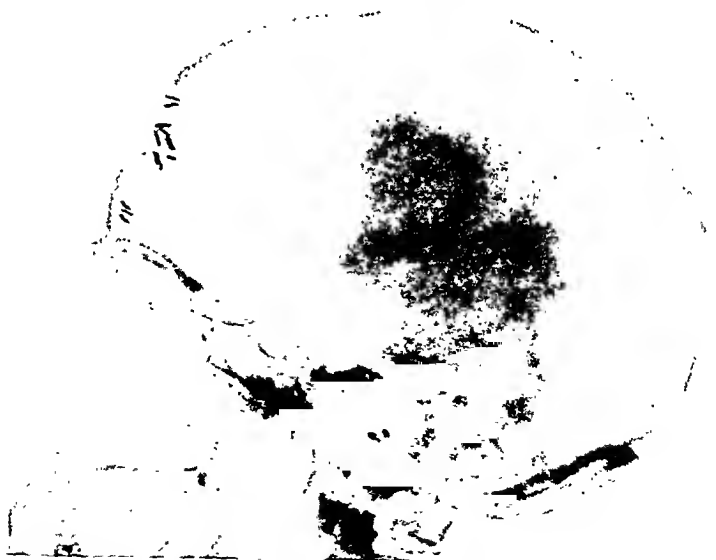


FIG. 427.—Right lateral skiagram, six days after second operation, showing complete removal of tumour and absence of air in ventricles.

back over it. On asking the patient to hold his nose and blow, it could be seen that the fascial implant elevated but remained *in situ*. When carrying out this

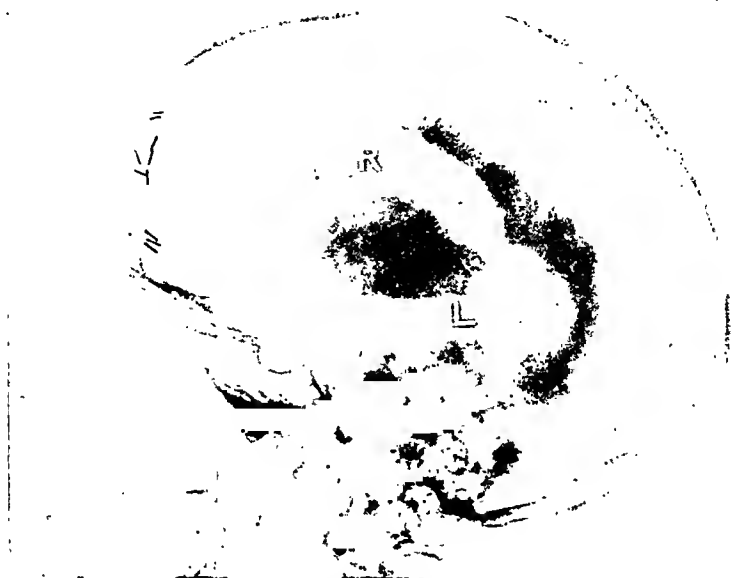


FIG. 428.—Condition found on April 22, 1930, after re-establishment of rhinorrhœa showing air in both right (R) and left (L) ventricles.

procedure it was also noted that a squeak could be heard which the patient said he had heard previously on occasion. This was found to originate in a very small

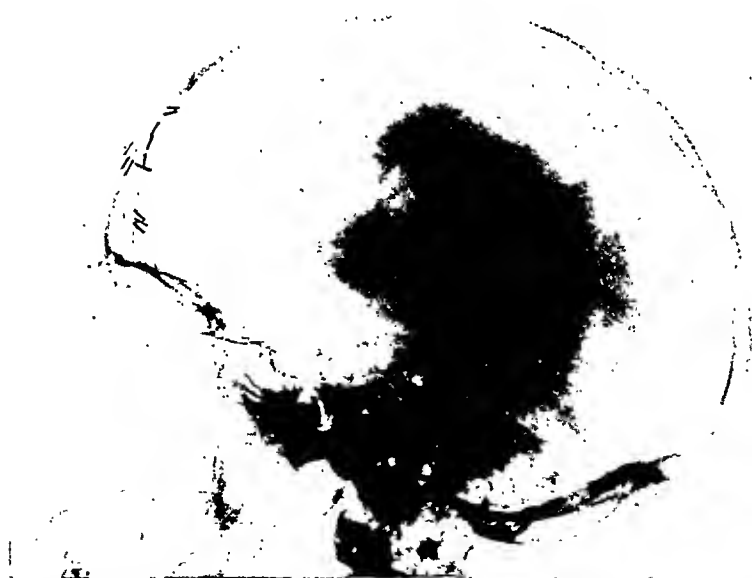


FIG. 429.—Right lateral radiogram taken on May 5, 1930, after the third operation, showing almost complete disappearance of air from the ventricles.

communication with the right anterior ethmoidal air-cells. Accordingly the mucous membrane was reflected therefrom, a fascial stamp applied, and the mucous membrane flaps were replaced over it. It was decided not to fill the cavity with salt solution but to leave it dry. Closure without drainage.

Post-operative Course.—The immediate post-operative condition was satisfactory, but on the third day the patient became extremely drowsy and could only be aroused with difficulty. The temperature had risen to 102° , the white-cell count being 10,400, and he was obviously mentally upset and very stuporous. On the fourth day, however, he commenced to improve, so much so that he was up in a wheel-chair on the eleventh day with no sign of cerebrospinal rhinorrhœa. X-ray examination on May 5 (*Fig. 429*) showed almost complete disappearance of air from the ventricles. There was no protrusion at the site of operation. His final condition can best be seen in the photographs taken on May 9 (*Figs. 430, 431*). X-ray



FIGS. 430, 431.—Patient on discharge from hospital, May 9, 1930.

examination carried out on this date showed that all the air inside the skull had been absorbed. He was discharged from hospital cured on May 15.

Three and a half months later, on Aug. 27, he returned for examination. He had been perfectly well, without any recurrence of cerebrospinal-fluid rhinorrhœa, had gained forty pounds in weight, and was working full time as an electrical supervisor. X-ray examination on this date showed no sign of any air inside the skull.

SUMMARY.

An historical survey of the subject of osteomata of the perinasal sinuses is given in association with a report of a large frontal osteoma complicated by multiple mucocœles, one of which communicated with the lateral ventricle, causing a cerebrospinal rhinorrhœa and a ventricular pneumatocele. The bony tumour was removed in two stages, and a third was necessary to close two cerebrospinal fistulæ by means of fascial implants.

I wish to express my indebtedness to Dr. Cushing for kind permission to publish the case, and also for his helpful criticisms and advice.

REFERENCES.

- ¹ CUSHING, *Surg. Gynecol. and Obst.*, 1927, xliv, 721.
- ² PARSONS, *Pathology of the Eye*, 1904, ii, 745.
- ³ PAGET, *Lectures on Surgical Pathology*, 3rd Amer. ed., 1865, 477, 478.
- ⁴ HOME, *Mcd. and Surg. Catalogue of Gen. Pathol.*, 1799, vii, 172.
- ⁵ MACKENZIE, *Practical Treatise on Diseases of the Eye*, 1830, 55.
- ⁶ BOUYER, *Ann. de la Chir. et étrang.*, 1841, iii, 242.
- ⁷ ROKITANSKY, *A Manual of Pathological Anatomy* (translated from the German by Swaine), 1854, i, 181.
- ⁸ WEISS, *Bull. Soc. anat. de Paris*, 1851, xxvi, 220.
- ⁹ CRUVEILHIER, *Traité d'Anat. pathol. gén.*, 1856, iii, 869.
- ¹⁰ VIRCHOW, *Die krankhaften Geschwülste*, 1865, ii, 37, 42.
- ¹¹ HILTON, *Guy's Hosp. Rep.*, 1836, i, 237.
- ¹² ROUX, *Mém. Acad. roy. de Méd.*, 1871, iii, 4.
- ¹³ DOLBEAU, *Bull. Acad. de Méd.*, 1871, xxxvi, 564, Plate II, Figs. 1-4.
- ¹⁴ KNAPP, *Graefe's Arch. f. Ophthalmol.*, 1861, viii, 239.
- ¹⁵ ARNOLD, *Virchow's Arch.*, 1873, lvii, 145.
- ¹⁶ BANGA, *Deut. Zeits. f. Chir.*, 1874, iv, 486.
- ¹⁷ LUCAS, *Edin. Med. and Surg. Jour.*, 1805, i, 405.
- ¹⁸ KNAPP, *Trans. Med. Soc. N. Y.*, 1881, 252.
- ¹⁹ BERLIN, *Graefe-Saemisch Handb. der Augenheilk.*, 1880, vi, 729.
- ²⁰ ANDREWS, *Med. Record*, 1887, xxxii, 261.
- ²¹ TAUBER, *Zentralb. f. Chir.*, 1898, xxix, 775.
- ²² BORNHAUPT, *Arch. f. klin. Chir.*, 1881, xxvi, 589.
- ²³ KNAPP, *Arch. of Ophthalmol.*, 1880, ix, 185.
- ²⁴ HUCKLENBROICH, *Inaug. Diss.*, Freiburg, 1905.
- ²⁵ GERBER, *Arch. internat. de Laryngol.*, 1907, xxiii, 1.
- ²⁶ SEVALL, *Ann. of Otol. Rhinol. and Laryngol.*, 1918, xxvii, 275.
- ²⁷ LEE, *Laryngoscope*, 1925, xxxv, 291.
- ²⁸ HARRIS, *Ibid.*, 1928, xxxviii, 331.
- ²⁹ BROWDER, *Arch. Otolaryngol.*, 1929, ix, 297.
- ³⁰ KIKUZI, *Beitr. z. klin. Chir.*, 1888, iii, 489.
- ³¹ CULBERT, *Ann. of Otol. Rhinol. and Laryngol.*, 1918, xxvii, 1203.
- ³² DUBAR, *Des Ostéomes des Fosses nasales et des Sinus voisins* (Thesis), 1900, Paris.
- ³³ DE TARANTO, *Les Ostéomes de l'Orbite*, 1901, Paris.
- ³⁴ MIODOWSKI, *Knochen Orbital Tumoren*, *Inaug. Diss.*, Breslau, 1900.
- ³⁵ AXENFELD, *Klin. Monats. f. Augenheilk.*, 1904, xlii, 229.
- ³⁶ WRAY, *Proc. Roy. Soc. Med.*, 1915, viii, 126.
- ³⁷ MACKENTY, *Ann. of Otol. Rhinol. and Laryngol.*, 1918, xxvii, 1116.
- ³⁸ BLAND-SUTTON, *Tumours, Malignant and Non-malignant*, 7th ed., 1922, 53.
- ³⁹ THOMAS, *Amer. Jour. Otolaryngol.*, 1918, v, 340.
- ⁴⁰ BARNHILL, *Ann. of Otol. Rhinol. and Laryngol.*, 1918, xxvii, 1239.
- ⁴¹ INGERSOLL, *Ibid.*, 1116.

TRANSPLANTATION OF AN EXTERNAL BILIARY FISTULOUS TRACT INTO THE DUODENUM.

By GEORGE E. WAUGH,

SENIOR SURGEON TO HAMSTEAD GENERAL HOSPITAL.

RECENTLY Waltman Walters¹ reported his own fifth case of transplantation of an external biliary fistula into the stomach or duodenum. At the end of his description he writes, "In the literature the total number of cases of transplantation of these fistulas, including the 5 which I have reported, is 12." In view of the rather surprisingly small number of cases that have been put on record, it may be worth while to give the details of one more case of an external biliary fistula which was transplanted successfully into the duodenum by the writer in February, 1929. Although the patient is 74 years old, he is still at the date of writing (October, 1930) in excellent health.

Many plastic operations upon the bile-duets are described in authoritative text-books of surgery such as Moynihan's *Abdominal Operations*, *The Art and Science of Surgery* by Mitchiner and Romanis, and *Surgical Operations* by Rowlands and Turner, but this particular variety seems to have escaped attention. Under the heading "External Biliary Fistula" the possible cause, such as retained calculus or stenosis of the common duet, is fully discussed, together with details of operative procedures which must be modified according to the condition actually found at operation; but transplantation of the whole fistulous tract is not mentioned. Perhaps the necessity of doing so is as uncommon as would appear from Walters's statement; or under certain pathological conditions it is so obviously the only thing that can be done that operators have not considered it worthy of record.

One aspect of this condition, however, seems to require special emphasis, and that is the necessity of preserving the whole of the fistulous tract intact until a preliminary dissection has revealed the exact pathological state of the biliary apparatus. In this particular case the history of the previous operation seemed to suggest that a portion of the gall-bladder combined with some obstruction of the common duet would be found; and with this pre-fixed idea it was more by good fortune than design that the whole tract was preserved in its integrity. If it had been cut across at any earlier stage of the operation to facilitate an arduous and difficult dissection, under the impression that parts of the gall-bladder and cystic duet were contained therein, the result would have been disastrous. Only when all the structures in the matted gastro-hepatic omentum had been dissected clean was it possible to realize that the whole of the common duet, common hepatic duet, cystic duet, and gall-bladder had disappeared, and that the now isolated tract led from a lateral opening in the right hepatic duet to the surface of the body (see Fig. 433). Walters in one of his cases made an artificial

external fistula by draining an "intra-hepatic duct to the surface", and two months later transplanted successfully the artificial tract into the stomach. The reason for doing this was that all traces of the normal bile-ducts had disappeared following upon a cholecystectomy done by some other surgeon, and the patient had suffered from complete obstructive jaundice for one month before coming under Walters's care.

HISTORY.—W. M. G. G., male, age 73 years, was first seen in February, 1929, in consultation with Dr. W. Jagger and Dr. Evelyn Rich. It was then stated that one year previously an emergency operation had been performed upon his gall-bladder and that the wound had never healed. The few details that were obtainable about the operation indicated that a gangrenous portion of the gall-bladder had been excised and a tube inserted into the remainder.

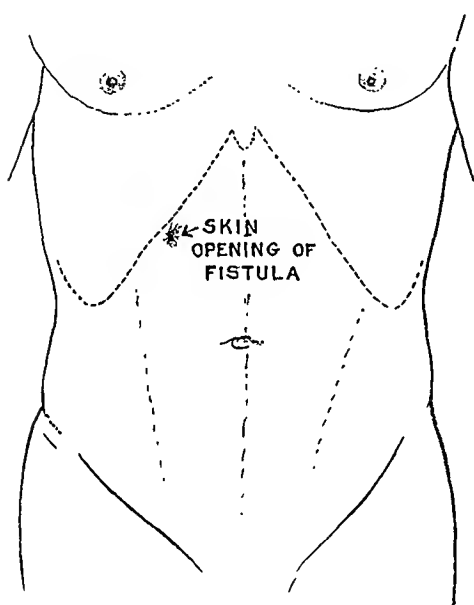


FIG. 432.—Showing site of external biliary fistula.

A complete biliary fistula had formed at the site of the drainage-tube (*Fig. 432*), and apparently the whole of the bile that was formed passed on to the surface of the body. The stools were clay-coloured; the urine contained only a faint trace of pigment; and the copious output of bile on the surface soaked through enormous dressings of wool three times daily. He had undergone an operation for the removal of his prostate ten months previously to the gall-bladder operation, and this had left him with permanent incontinence of urine. With two external fistulas his plight was very miserable, and although he was a high operative risk it was decided to attempt the closure of the biliary fistula. In addition to the handicap of his age he had emphysema and a slight tendency to bronchitis.

OPERATION (Feb. 25, 1929).—Gas and oxygen were administered by Dr. F. A. Seymour, and the lower six intercostal nerves on both sides were infiltrated with 1 per cent solution of novocain and adrenalin just anterior to the angles of the ribs. The opening of the abdominal cavity, after having turned the upper two-thirds of the right rectus outwards, was a matter of extreme difficulty on account of dense adhesions matting intestines to one another and to the anterior abdominal wall. After a prolonged dissection, during which the fistulous tract was isolated, the site of the gastro-hepatic omentum was defined ultimately. The foramen of Winslow was closed by stout fibrous tissue, and the whole of the gastro-hepatic omentum in the immediate neighbourhood was an equally stout fibrous mass from which the inner end of the fistulous tract emerged just below the transverse fissure of the liver. This mass was opened up cautiously from the upper border of the

duodenum to the level of the tract, and the portal vein and hepatic artery were stripped clean. The tract thus defined was then seen to emerge from the outer margin of the right hepatic duct (*Fig. 433*), whilst below that point all other portions of the biliary apparatus were absent. The surface opening of the fistula was detached from the skin, and, after mobilizing the second part of the duodenum, an easy anastomosis was made between the two through an incision in the duodenum, with two layers of continuous catgut sutures applied in the manner of an ordinary gastrojejunostomy operation

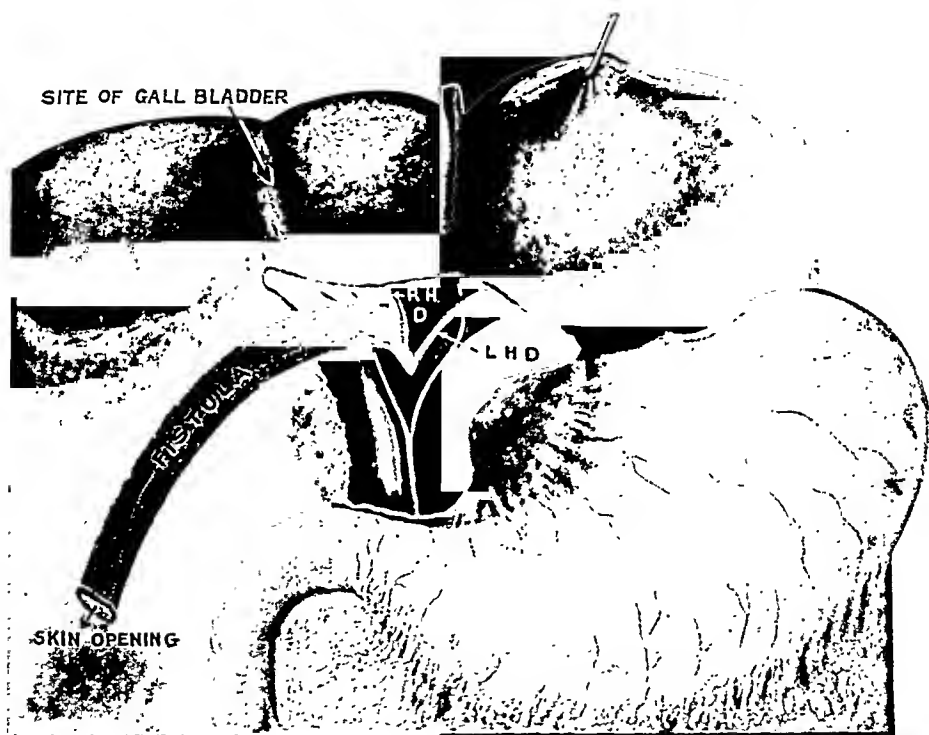


FIG. 433.—The liver has been turned upwards and the area of dissection in the gastro-hepatic omentum is shown outlined by a white line. The relative sizes of the fistulous tract and the hepatic ducts have been drawn to scale.

(*Fig. 434*). Omentum was stitched to the site of the anastomosis and a tube inserted down to it through the old fistulous opening on the skin. This proved to be superfluous and was removed at the end of forty-eight hours. The total duration of anæsthesia was two and a half hours, and $2\frac{3}{4}$ oz. of ether were administered at one stage to improve relaxation. The pulse-rate was only 80 at the end of the operation, and had fallen to its normal rate of 68 on the following day.

SUBSEQUENT PROGRESS.—The convalescence was remarkably smooth. For a full period of seventy-two hours the patient was allowed nothing

except abundant subcutaneous infusions of saline solution, 20 per cent glucose and saline per rectum, and hypodermic injections of heroin to ensure rest and sleep. At the end of this period raw eggs beaten up with glucose and brandy were given by mouth. The bowels acted of their own accord on the fifth day, and some pigment was present in the stool. Subsequent motions were normal in colour and the urine became a deeper yellow. He went home at the end of four weeks from the day of operation.

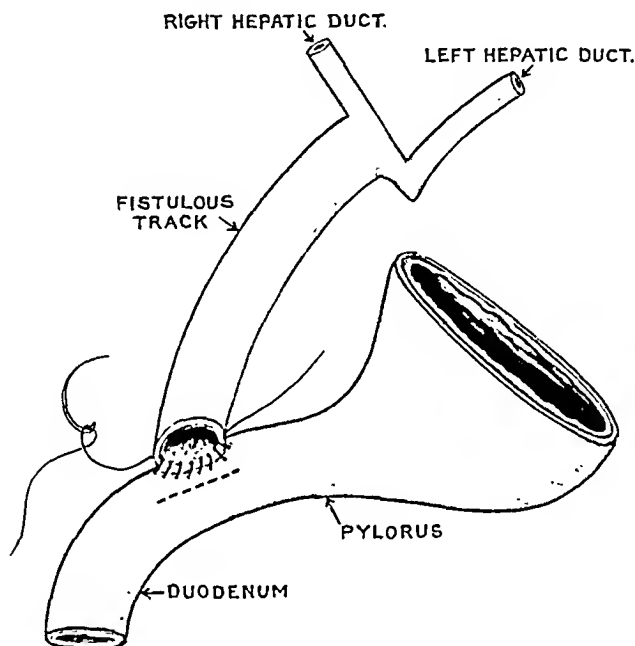


FIG. 434.—The method of anastomosis is shown in the first stage; the dotted line on the duodenum marks the line of incision into it necessary to complete the second stage.

On Oct. 5, 1930, Dr. E. Rich reports. "G. G. is wonderfully well except for his incontinence of urine".

The illustrations were drawn by Mr. S. A. Sewell from rough sketches of my own made at the time of operation.

REFERENCE.

- ¹ *Proc. Staff Meetings of the Mayo Clinic*, 1930, May, v, No. 21.

UNUSUAL COMPLICATION ARISING DURING OPERATION FOR A LARGE HYDATID CYST OF THE LIVER.

By JAMES A. JENKINS, DUNEDIN, N.Z.

THE following case appears to be worth recording, as I am unable to find in the literature available any report of a similar complication arising during operation.

The patient, a male, age 43, married, was admitted on May 14, 1930, complaining of epigastric pain and jaundice of ten days' duration.

PREVIOUS HISTORY.—No previous illnesses. Does heavy manual work. Habits satisfactory. Nothing of importance in the family history.

PRESENT ILLNESS.—When at work ten days previously, he was suddenly seized with severe epigastric pain. It gradually eased off and he carried on with his work for that day. He took salts on arrival at his home and vomited shortly after. The next day he was jaundiced. He remained at home for a few days, and then returned to work, but still felt ill. The jaundice progressed, and on the day before admission he had another attack of severe pain which radiated to the back. He was sent into hospital with a diagnosis of cholecystitis.

ON ADMISSION.—Temperature 100.5° , pulse 75, respirations 25. He was deeply jaundiced, the tongue was coated, and he looked a rather sick man. The edge of the liver could not be felt, but there was an ill-defined rounded fullness of the right lobe, extending below the right costal margin for about two inches on deep respiration. Bile was present in the urine, and the stool was clay-coloured. Other systems revealed no abnormality worth recording. X rays showed the right dome of the diaphragm to be much raised, and that general enlargement of the liver was present. The stomach was shown by barium meal to be pushed over to the left. Tests: Kahn and Wassermann, negative; Kolmer quantitative (hydatid), + + + + +; hydatid complement fixation, strongly positive; Casoni, strongly positive; van den Bergh—qualitative, immediate direct; quantitative, 5.5 units; T.N.P.N., 34 mgrm. per 100 c.c.

Calcium chloride was given intravenously, and blood coagulation time on the day of operation was normal. His temperature during the six days he was in the medical ward prior to operation ranged from 99.9° to 100° . His jaundice progressed, and the stools remained clay-coloured throughout. He was transferred for operation on May 20 with the diagnosis of hydatid of the liver, and we considered daughter cysts in the common bile-duct to be the most likely cause of his jaundice.

OPERATION.—Anæsthetic: ether combined with local nerve-block of abdominal wall. Right paramedian incision with reflection outwards of the right rectus muscle. The right lobe of the liver was somewhat enlarged and

the normal contour was lost. The omentum was adherent to the diaphragmatic surface of the liver. On palpation it felt very firm, tense, and unyielding. No fluctuation was present. On the diaphragmatic surface of the liver there was considerable fibrous change. The left lobe had undergone marked hypertrophy, presumably of a compensatory nature. The gall-bladder was tense and distended. Examination of the region of the portal fissure and common bile-duct showed that the large cyst present was not responsible for the obstruction of the common bile-duct. The head of the pancreas felt normal. No attempt was made to explore the common bile-duct, as the patient was too ill to permit of any non-essential prolongation of the operation. The enlarged liver made the common bile-duct very difficult of access. The original diagnosis of daughter cyst in the common bile-duct was considered highly probable, and cholecystostomy was performed with the view to removal of the daughter cyst at a second stage.

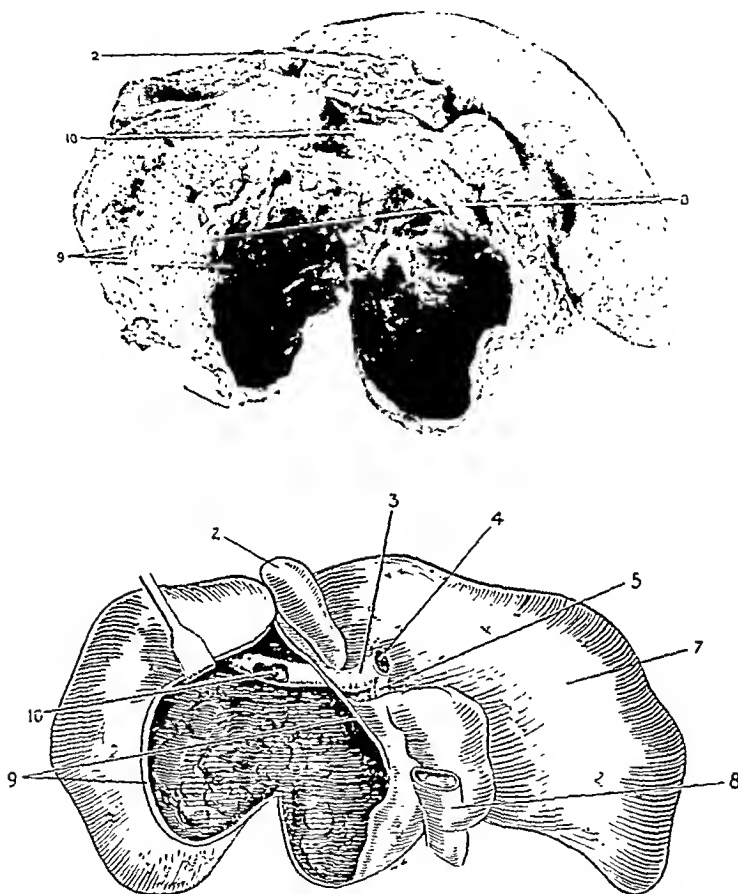
The cartilaginous costal margin was rapidly resected, and the presenting surface of the liver sutured to the parietal wound. This was readily done in view of the fibrous change in this part of the liver. There was no hæmorrhage. The lower abdominal wound was closed up to this point, thus isolating the field of potential infection. An area of about 3 in. by 2 in. of presenting cyst was packed off, the cyst tapped and formalinized, and then incised. About a third of an inch of tough fibrosed liver (pericyst) had to be cut through before entering the cyst. Daughter cysts came away freely, and their expulsion was aided by gentle irrigation through a $\frac{3}{4}$ -in. glass tube. In all, two to three quarts of cyst and fluid were expelled without any manipulation of the interior of the cyst other than irrigation, when venous oozing commenced. At first it was not very severe, but in a few minutes it assumed alarming proportions. It welled up from the depth of the cyst, and in the course of two or three minutes the patient's condition changed from one of comparative safety to one causing the gravest concern.

The site and source of the hæmorrhage was not known. I realized that a large vein had ruptured into the cyst cavity after the tension had been reduced, but whether a branch of the portal vein or vena cava I did not know. The abdomen had been closed prior to opening the cyst, hydatid daughter cysts were presenting every second or so, and at first I was loath to re-open the abdomen and compress the portal vein and hepatic artery in the lesser omentum. At the end of about two minutes the patient's condition was such that this procedure would probably have precipitated death on the table.

The cyst cavity was filled up with saline to which was added a little flavine. The opening into the cyst was very tightly plugged with gauze (tight suturing would have been much better), the wound packed, and the patient returned to bed. One pint of blood, two of intravenous saline, followed by a further pint of blood, were given. Death occurred a few hours later from continued hæmorrhage into the cyst cavity.

POST-MORTEM.—Autopsy revealed the source of the hæmorrhage. The right branch of the portal vein, immediately after entering the liver, passed through the pericyst (fibrous condensation of tissue surrounding the hydatid), and its wall was completely eroded (*Figs. 435, 436*). The index finger could

be passed from the cavity into the vein. Some old thrombus was present at this site. The whole of the right lobe of the liver was destroyed, being expanded over the cyst. The left lobe was greatly hypertrophied. In the common bile-duct near the ampulla a daughter cyst was tightly lodged.



FIGS. 435, 436.—Under surface of liver, showing right lobe largely converted into a cyst with dense leathery pericyst. Inside the cyst are seen daughter cysts and membranes of the mother cyst, and, embedded in the upper part of the anterior wall, the right branch of the portal vein is seen, with a large erosion which permitted the fatal hæmorrhage. The left lobe shows marked compensatory hypertrophy. 2, Gall-bladder; 3, Right branch of portal vein; 4, Portal vein; 5, Left branch of portal vein; 7, Hypertrophied left lobe of liver; 8, Vena cava; 9, Divided wall of cyst; 10, Right branch of portal vein passing through the pericyst, showing erosion.

COMMENTARY.

Biliary colic and jaundice of an obstructive type due to daughter cysts or débris in the ducts have been so frequently reported in Australasian publications that discussion of this aspect of the case, though of interest, does not differ from many already published. Its frequency is such that a

provisional diagnosis of daughter cyst in the common bile-duct was made prior to operation.

Profuse hæmorrhage may occur from large veins should it be necessary to cut through liver tissue, and also if misplaced attempts are made to remove calcareous pericyst. That danger is always present in large cysts is obvious when the close association of portal vein and vena cava is appreciated. Spontaneous rupture of cysts into the vena cava or its tributaries accounts for sudden death from parasitic emboli or anaphylaxis (Pitts,¹ Bird²). The reverse procedure, or rupture of an eroded vein into the cavity within the pericyst, after the support of the hydatid membrane is withdrawn, is almost unique. Syme³ reported a terrific hæmorrhage from an erosion of the inferior vena cava by the cyst. His case is the only one I can find reported in the literature in any way parallel to the one here discussed.

Procedures adopted in the treatment of large cysts of the liver vary in detail, but the two essentials are: (1) To avoid contamination of wound, peritoneum, pleura, etc.; (2) To eradicate the endocyst, ectocyst, and cyst contents. A large cyst usually presents near the surface of the liver at some part of its circumference, but the area of thinned liver tissue is very often not in an area readily approached surgically, or suitable for treatment after the cyst is emptied. Abdominal approach permits of a general survey of liver and other abdominal contents at operation, and secondary cysts and other complications are more readily dealt with; but if the cyst is presenting under the right cupola of the diaphragm, approach after rib resection is most commonly adopted. Whether infected with pyogenic organisms or not, scrupulous attention must be paid to isolation of the field of operation before the cyst is opened. A two-stage operation, to permit of scaling off, is for this reason often to be recommended. After the cyst is exposed and the field effectively sealed and packed off, it is formalinized in order to kill scolices. To do this the cyst is aspirated and the contents replaced with 1 per cent formalin. That formalin is a very destructive agent on the host's tissues is well known, and should it get in contact with pericyst outside the cyst membrane and remain there for any length of time, gross destruction of tissue may occur later. In a cyst packed with daughter cysts the likelihood of efficient sterilization is not great whatever method is adopted. Dew⁴ recommends the injection of a small quantity of pure formalin so as to make a final solution of 1 per cent. The required amount is hard to estimate in any individual case. After removal of cyst contents, the membranes of the mother cyst usually separate spontaneously. Gentle irrigation of the cavity with saline washes out all formalin and brings fresh daughter cysts to view. Other cysts may then be seen bulging into the one already dealt with.

All liver hydatids are potentially infected. Biliary fistula is very common, and it is this liability to infection after operation that makes marsupialization, with or without tube drainage, the procedure so commonly adopted. The large cavity takes considerable time to become obliterated by falling in of its walls and the growth of granulation tissue. Secondary infection with its attending evils takes its toll if drainage is done. In cavities presumably sterile (though there is always the risk of the entry of bile, with resulting low-grade infection), one may adopt the method of filling the cavity

with normal saline followed by closure of the cyst. Russell⁵ adopted this method with success in infected cysts. Gross infection is an indication for drainage. An intermediate course is probably the safest procedure. The cyst is filled with saline to which some non-irritating antiseptic such as flavine may be added, and is closed tightly. The line of suture is then attached to the peritoneal opening, or a tube is carried down to the vicinity of the cyst. Should suppuration occur, there will be spontaneous drainage, or, if this fails, a tube may be readily inserted.

Calcification of the pericyst is always troublesome, and attempts at removal usually result in severe hæmorrhage. Recurrent abscesses and persistent sinuses are often present, necessitating a lengthy convalescence.

The grossly infected hydatid demands free drainage and the same general and local surgical measures required in any abscess cavity of this organ.

I wish to express my thanks to the Pathological Department of the University of Otago Medical School for the preparation of the specimen and photographs, and to Sir Louis Barnett for much help and advice.

REFERENCES.

- ¹ PITTS, A. B., *N.Z. Med. Jour.*, 1919, Aug., 191.
- ² BIRD, *Med. Jour. of Australia*, 1925, March 14, 258.
- ³ SYME, SIR GEORGE, *Ibid.* (Suppl.), 1927, Nov. 19, 393.
- ⁴ DEW, *Hydatid Disease*.
- ⁵ RUSSELL, H., *Austral. Med. Gaz.*, 1907, Oct. 21, 514.

RENAL CALCULI WITH SQUAMOUS CARCINOMA IN A HYDRONEPHROTIC KIDNEY.

By L. WOODHOUSE PRICE,

PATHOLOGIST TO THE GLASGOW ROYAL CANCER HOSPITAL;

AND ARTHUR JACOBS,

UROLOGIST TO THE GLASGOW ROYAL INFIRMARY.

A CASE of epithelioma occurring in a horseshoe kidney with giant renal calculus has been described by Willan.¹ The following case seems worthy of being placed on record as another example of the somewhat rare occurrence of primary squamous carcinoma in this situation.



FIG. 437.—Hemisection of removed kidney, showing extensive cystic dilatation, with multiple calculi and the tumour in the lower segment.

HISTORY.—The patient, a labourer of 54, was referred to the Urological Clinic at the Glasgow Royal Infirmary complaining of left lumbar pain. This had been present practically without intermission for a year. There were periods when the pains became exacerbated and radiated to the left testicle. He also complained of inability to keep his food down, and stated that, as a

result, he had lost two stone in the last few months. Although he apparently considered that his complaint was only of one year's duration, he admitted that he had been subjected to intermittent attacks of discomfort in the left renal area for a period extending over eighteen years. He did not suffer from urinary frequency or dysuria, though he had noticed that his urine was 'muddy'.

ON EXAMINATION.—A large mass could be felt in the left abdomen, extending from under the left costal margin down to the iliac region. The urine contained albumin, blood, and pus. A radiographic examination showed shadows of at least eleven calculi of varying dimensions occupying an area extending from below the iliac crest upwards to the level of the 2nd lumbar vertebra. An intravenous pyclogram adequately demonstrated

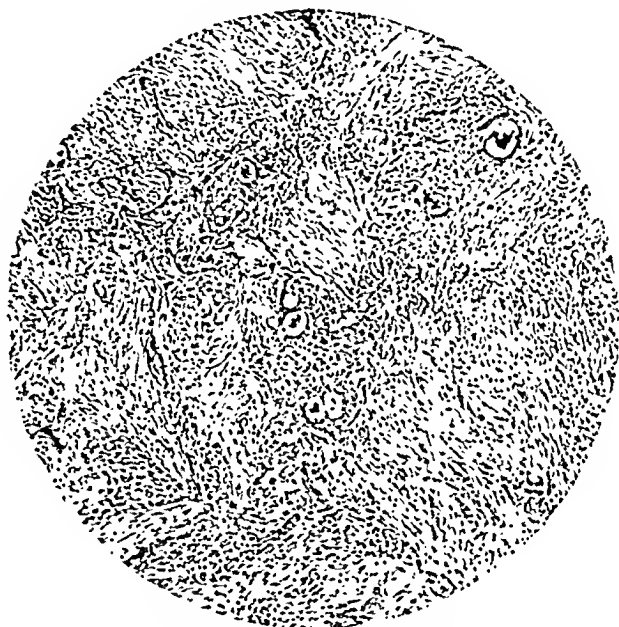


FIG. 438.—Infiltration of the fibrotic renal parenchyma with irregular masses of squamous carcinoma. ($\times 60$.)

a normal renal pelvis on the right side and a very gross hydronephrosis on the left, fifteen minutes after the injection of 40 gm. of uroselectan.

OPERATION.—The kidney was exposed through a curved lumbar extra-peritoneal incision. Although the external arcuate ligament was cut and the incision prolonged forwards to beyond the level of the anterior superior spine, great difficulty was experienced in attempting to deliver the organ on account of its size. After resecting the twelfth rib, however, this was successfully accomplished. Following on division of the ureter and ligation of the pedicle the kidney (*Fig. 437*) was removed without further trouble. A rubber drainage-tube was inserted at the upper angle of the wound and the incision closed.

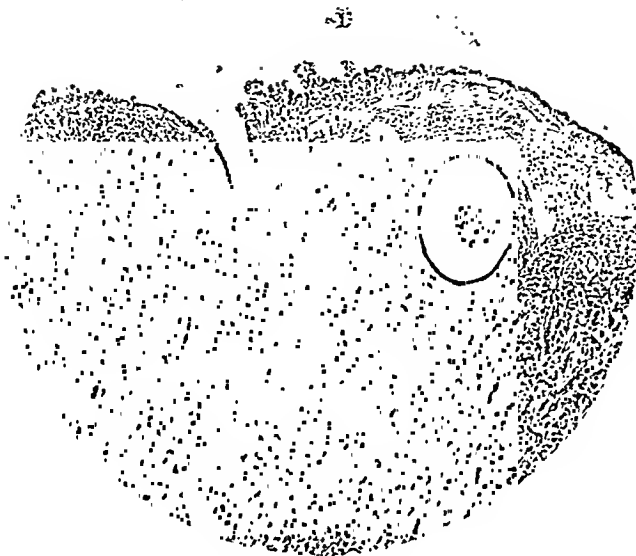


FIG. 439.—Ulceration of the transitional epithelium of the renal pelvis from irritation by calculi, and columns of malignant cells infiltrating the fibrotic renal parenchyma. ($\times 60$).



FIG. 440.—Transitional epithelium lining a dilated calyx in the neighbourhood of the tumour, showing a tendency to undergo hyperplasia. The underlying renal tissue shows chronic inflammatory change. ($\times 60$).

The patient left the operation theatre in good condition, but a few hours later exhibited symptoms of severe shock which lasted until the following day. There followed an uninterrupted convalescence, and he left hospital twenty-one days after operation.

PATHOLOGICAL ANATOMY.—The kidney was enlarged, measuring $7\frac{1}{2}$ in. from pole to pole and $4\frac{1}{2}$ in. in its greatest transverse diameter. The surface was coarsely lobulated, and the organ was obviously cystic and contained fluid. There was a considerable amount of perirenal fat, especially around the pelvis of the organ. On dissection the pelvis and first inch of the ureter were found to be obstructed by a large calculus of the 'stag-horn' type which was firmly adherent to the lining membrane of the ureter. The pelvis, ureter, and neighbouring renal tissue were thickened and indurated. The surface of the organ showed a number of punctate hæmorrhages, most marked in the region of the lower pole. While the greater part of the organ appeared cystic, the lower pole showed a solid mass of tissue, almost white in colour, projecting as a nodule the size of a walnut towards its posterior aspect. This mass appeared to be completely separated from the pelvis by cystic renal parenchyma.

On hemisection of the organ the renal parenchyma was found to be practically obliterated, being reduced to a thin shell of tissue not more than 1 mm. in thickness. The interior of the organ was occupied by large cystic spaces containing fluid, separated from each other to a greater or lesser extent by greatly attenuated renal substance. The region normally occupied by the pyramids contained a quantity of dense fibro-fatty tissue, partly embedded in which were a number of large irregular calculi. The normal configuration of the kidney could not be distinguished. At the lower pole the dilated calix was partly occupied by a cauliflower-like tumour about the size of a large walnut. In the centre of the tumour mass were two irregular spaces occupied by a number of small faceted calculi. Distributed irregularly in the fibro-fatty tissue of the central portion of the organ, but occupying chiefly the lower half, were several large calculi.

HISTOLOGY.—Sections prepared from tissue removed from the tumour occupying the lower pole of the kidney showed the structure of a squamous-celled carcinoma. This presumably arose in the epithelium of the lower calix, which had undergone squamous metaplasia, as the transitional epithelium in this region showed hyperplasia and in places a definite tendency to assume squamous characteristics. The epithelium of the pelvis and upper end of the ureter, on the other hand, though ulcerated from the presence of calculi, showed no evidence of hyperplasia or of squamous metaplasia. The accompanying microphotographs (*Figs. 438-440*) illustrate the histology of the neoplasm and of the diseased renal parenchyma.

REFERENCE.

- ¹ WILLAN, R. J., "Giant Renal Calculus with Epithelioma in a Horseshoe Kidney", *Brit. Jour. Surg.*, 1928, xvi, 317.

CONCERNING THE PATHOLOGY OF TUMOURS OF TENDON-SHEATHS.

By E. S. J. KING,

STEWART LECTURER IN PATHOLOGY AT THE UNIVERSITY OF MELBOURNE.

THE great majority of the reports and descriptions of tendon-sheath tumours emphasize the yellow colour of the growths, the presence of giant cells, or other incidental features. In recent years an attempt has been made to express the characteristics of tumours and their relationship to cells in various parts of the body by the use of terms which indicate the nature of the cells comprising the growth, such as 'hepatoma', 'luteoma', 'meningioma', etc. The same idea has been applied to synovial membranes by the use of the term 'synovioma'. This has resulted from the realization that the potentialities of tumours must depend on the nature of the cells from which the growths arise and of which they are composed. An appreciation of these growths can be achieved only by way of normal and abnormal synovial membranes.

HISTORICAL.

A continuous synovial lining was described first in 1763 by Bonn, and in 1800 Bichat showed that the 'Haversian glands' were not mucus-producing but consisted of fat. Originally joints were thought to be lined by epithelium, but the descriptions by His and Remak of the developmental relations of the three germinal layers led to this hypothesis being discarded. It was replaced by the idea of an 'endothelial' lining—a view championed by Schweigger-Seidel, Landzert, Steinberg, Colomiotti, Bentzen, and Tillmans.

In 1866 Hueter, by using a silver impregnation method, came to the conclusion that the lining of joints was not continuous, but incomplete and irregular. His view was upheld by Reyher, van der Sluijs, Hagen-Torn, Hammar, Braun, and Tourneux and Herrmann. A modification of Hueter's view, that the synovial membrane is a specialized connective-tissue surface, was introduced in 1910 by Lubosch. This was supported by Segale, and has now received ample confirmation. It is astonishing that most discussions on the subject of joint or tendon-sheath synovial membranes disregard completely all this work and refer to 'endothelial linings'. It is proposed to demonstrate that the nature of the tumours depends directly on the normal histological character of the tissue of the tendon-sheaths.

The first example of a tendon-sheath tumour was reported in 1852 by Chassaignac. This description was followed by others—Spencer Wells (1857), Billroth, Czerny (1868), and others—until 1882, when, in addition to clinical and macroscopic examination, histological investigations were undertaken.

Tourneux, who gives a very full account of the literature, describes this as the period of clinical observations. His second or histological period includes observations made by Zwiecke (1882), Markoe (1884), and Reverdin (1885) amongst others. The end of this period was in 1891—marked by the work of Heurtaux. From this time onwards there have been numerous observations and a number of attempts to classify these growths. In 1892 Dor described the xanthoma cells occurring in these tumours. In 1901 Bellamy, writing from Professor Stilling's laboratory in Lausanne, discussed 5 examples and concluded that the growths should be classed with the endo-theliomata. Rosenthal in 1909 discussed 71 cases, and in 1913 Tourneux referred to 93 cases. In 1915 Stewart and Flint reported 2 cases and, reviewing the literature, referred to 9 occurring before 1913 which had been overlooked by Tourneux. Fleissig suggested that many of them—the giant-celled tumours—were not neoplastic, but the result of a chronic inflammatory process. From this time onwards there have been only a few examples reported. Fourteen cases from the Mayo Clinic were described by Broders in 1919.

One of the striking features of all the reports and discussions is that most attention has been directed towards the presence of the foam cells (xanthoma cells) or the giant cells. Few attempts at histogenetic investigation have been made. In 1923 Buxton gave a classification of the growths according to the cell type, but did not indicate the relationships of these to each other or to the normal cells of the part. In 1928 Janik gave a more complete classification, but his work is open to the same criticism.

CLINICAL CHARACTERS.

The tendon-sheath tumours are insidious in onset. There is seldom pain, and the most constant observation is that of a swelling in the region of a tendon, most frequently in the hand. When first noticed the swelling may be small, or it may reach the size of a small orange before the patient seeks attention. It may be localized or may extend along the tendon. They usually grow very slowly, thus accounting for the absence of disturbance of the surrounding tissue.

Trauma is an important feature, particularly in the cases which show giant cells and foamy cells. The relationship is shown by the observation that such a large number of the growths occur on the flexor tendons of the right hand. Where the tumour was situated on the left hand, in the case described by Malherbe, the patient proved to be left-handed. The tumours are usually rounded in shape, are frequently lobulated, and possess a smooth surface. They vary considerably in consistence. In the early stages the growth does not interfere with movements, but later, either because it occurs at the region of a joint or because it invades it, function may be disturbed. The majority of the tumours are innocent, but a small proportion are malignant—some of them extremely so. Inadequate removal is liable to be followed by recurrence. If the tumour is near a muscle, this is invaded first, and later the other tissues are involved. In non-muscular regions the surrounding tissue is involved first, and later the tendon is itself affected.

When the skin becomes involved by the tumour, it becomes bluish and finally breaks down and the neoplasm fungates (*Figs. 441, 442*). (Czerny, Leclerc, and Bolognesi.) Infection of the tumour and subcutaneous tissues with sloughing occurs. Metastases are uncommon. They may occur in the regional lymph-glands (Bolognesi, Gaudiani, Leclerc, and Morestin). Visceral metastases are rare, but have been reported in the liver and lungs.

MACROSCOPIC APPEARANCES.

The tumours of tendon-sheaths are reddish-yellow, yellow, or grey in colour. They are attached to the sheath and may involve it for a considerable portion of its length on one aspect of the tendon, or may completely surround the tendon. At other times they are attached to the sheath

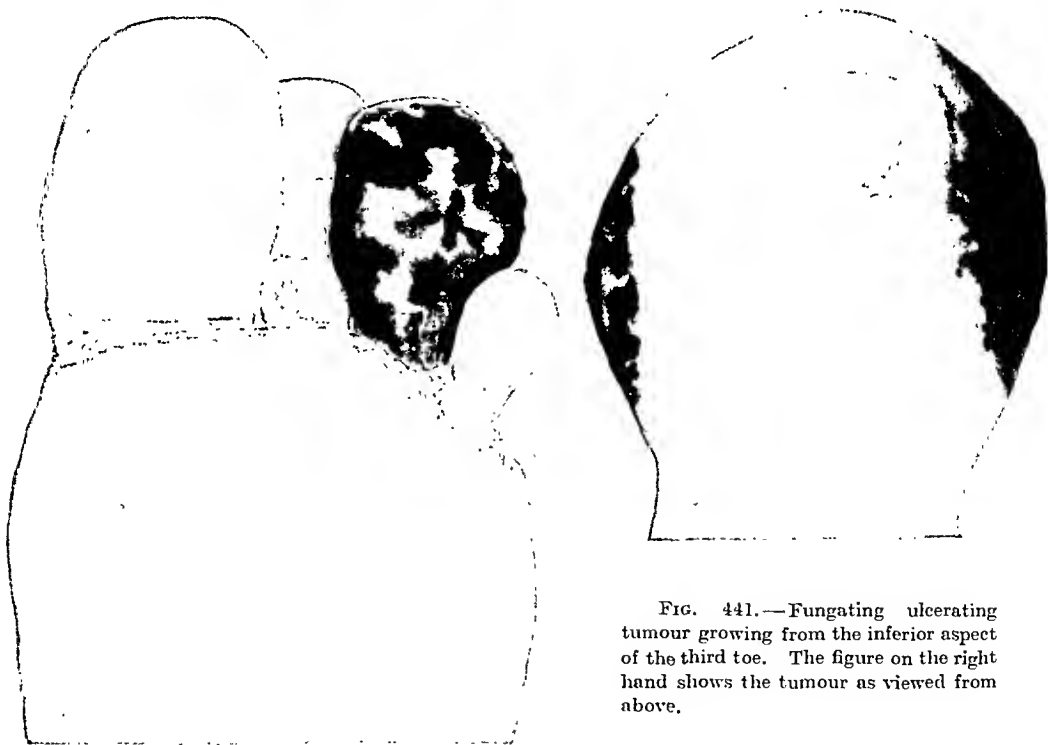


FIG. 441.—Fungating ulcerating tumour growing from the inferior aspect of the third toe. The figure on the right hand shows the tumour as viewed from above.

by a narrow pedicle. Their consistence varies with the nature of the growth. A very cellular growth is soft, and an admixture of fibrous tissue gives a firmness which varies with its amount. Calcareous material was observed by Billroth and by Czerny, and cartilaginous fragments by Heller. A complete chondroma was reported by Brenckmann, Buxton, Chauvain and Roux, Delbet, and Ombrédanne. A fibroma has been described by Buxton, Sandler, Sprengel, and Torek amongst others. The growths may be fatty

in both macroscopic and microscopic appearance (Meyer, Sendler, Sprengel, and others). Angioma and lymphangio-endothelioma (Faldini) have been reported.

On section, cellular, rapidly growing varieties are soft and of a uniform colour, and have an indefinite margin. Less rapidly growing forms are less



FIG. 442.—Microscopical section of the tumour shown in Fig. 441. The growth has invaded the tendon and also the overlying skin. 'Synovial' spaces are present throughout the substance of the tumour.

vascular, harder in consistence, and more definitely delimited. Some tumours resemble examples of chronic tenosynovitis, and it may be possible to distinguish them only after microscopic examination, e.g., by the presence of a greater number of mitotic figures than would be expected in inflammatory lesions (*see* Fig. 477).

MICROSCOPIC APPEARANCES.

Benign Varieties.—The innocent forms consist of variable proportions of spindle and spheroidal cells.

The Spindle Cells are somewhat variable in shape, showing an irregular, indefinite outline with well-marked protoplasmic processes. Sometimes one

end is expanded and the other is pointed. They resemble fibroblasts of various ages, and show all gradations into spheroidal cells. The protoplasm stains lightly with the counterstain and is homogeneous. There are sometimes present a few vacuoles of small size. The nucleus varies from an elongated to an oval form. It may be comma-shaped or triangular, etc. Some are small and pyknotic, while others are larger with an openwork chromatin net. One or two chromatin knots are present. Occasional mitotic figures are also present.

In parts the cells are closely placed with but few intercellular fibrils, but in other places there is a considerable amount of fibrous tissue. In some of the tumours (the so-called xanthomata and giant-cell tumours) there are many foamy cells, and in parts they may predominate. Giant cells may be present in considerable numbers. (*See Fig. 444.*)

The foamy cells are spheroidal in shape and contain a large number of fat-droplets, presenting a characteristic appearance. The true cells of the neoplasm, however, may be seen between these cells (*Fig. 446*), even where the foamy cells predominate. (*See Figs. 456, 457.*) In *Fig. 457* the tumour cells lining the space are included in the section.

Giant cells are of the foreign-body type (*Fig. 445*). They possess a considerable amount of granular protoplasm; the nuclei are small and similar to those of endothelial cells. Usually the nuclei are scattered irregularly through the protoplasm.

In many tumours some cells develop capsules, lose their processes, and resemble cartilage. Typical cartilage may be found and may comprise the whole tumour. Areas of mucoid change are frequent and are of two types. The first variety is the typical mucoid connective tissue seen in many connective-tissue tumours, particularly of the osteogenic type. This form has not been extensive in any of the growths examined by the writer. The second variety will be discussed later.

True fat tissue, as distinct from the foamy cells, occurs, and may predominate the picture, forming a 'lipoma' as described by Billroth, Mannini, Sandler, Sprengel, Strauss, and Tichoff, etc. It may occur as a papillary projection into the tendon-sheath space, giving rise to the lipoma arborescens, or it may be sessile. Bone occurs in some cases, and mixed tumours containing cartilage, bone, and mucoid tissue, such as the excellent examples described by Janik, may be found.

The Spheroidal Cells are arranged in more or less well-defined groups (*Fig. 447*). The cells are closely packed, and polygonal in shape (*Fig. 449*). There is a fairly definite cell outline. The protoplasm is lightly staining, non-granular, and vacuolated. The nuclei are rounded, oval, or sometimes indented, possessing a fine chromatin network. Some of the nuclei are particularly large. Occasional mitotic figures are present (*see Fig. 477*). Some of the cells are multinucleated.

In many parts this tissue merges into an acellular mucoid material (*Fig. 448*). The cells become fewer and larger. The cell walls become more definite and the nuclei smaller and pyknotic. The protoplasm undergoes concomitant changes, a number of large non-staining vacuoles forming.



FIG. 443.—Photomicrograph of a section of a tumour removed from the thumb. The tissue lining the space resembles that of synovial membrane, and is of the fibrous type. (Cf. Figs. 456 and 457.) ($\times 80$.)



FIG. 444.—Photomicrograph of another portion of the growth shown in Fig. 443. Giant cells and foam cells are present. ($\times 80$.)

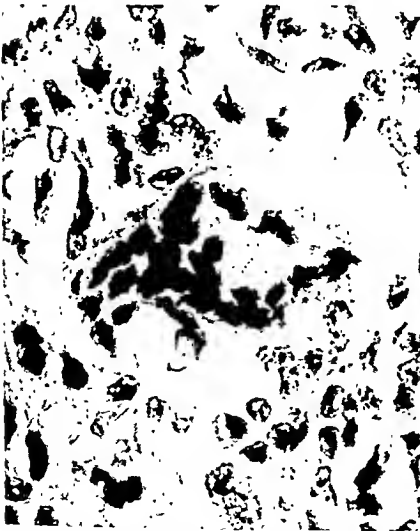


FIG. 445.—A higher power view of one of the giant cells in an area similar to that shown in Fig. 444. ($\times 540$.)



FIG. 446.—Another portion of the tumour shown in Fig. 443. Lipoid-containing cells are merging into fibrous tissue. ($\times 80$.)

Homogeneous intercellular material appears between the cells until only mucoid material containing some débris consisting of fragmented nuclei and some vacuolated protoplasm is present to the exclusion of all organized tissue. The material stains bluish with hæmatoxylin. These changes are very similar to those observed in ganglion formation.

Throughout all the tumours definite spaces occur amongst the cells (*Fig. 443*). These vary in size from small cavities little larger than the neighbouring blood-vessels to spaces occupying in length one or two low-power fields. These spaces occur in tissue of all types. They are lined by some of the tumour cells. The cells are surrounded by collagenous material, and though some are on the surface, others are just below the surface sending processes to it, and others are completely submerged. (Cf. *Figs. 453* and *469*.) These cells are continuous with the deeper cells of the tumour. They may be spindle (*Fig. 450*) or spheroidal cells (*Fig. 459*), or they may be fat-containing (*Fig. 456*). In many sections the cavities may be few and far apart, so that some areas are solid (*Fig. 446*).

Papillary growths occur into some of the cavities and resemble the papillæ seen in chronic tenosynovitis. These papillæ in some cases contain small round cells (*see Fig. 451*), indicating an inflammatory element in these growths. There are numerous droplets of various sizes in the protoplasm, suggesting a secretion. The vessels are well formed and are distinguishable from the tissue spaces mentioned.

Malignant Varieties.—The malignant forms of tumours may be interpreted when the innocent tumours have been studied. Most of the features are reproduced. The cells are of the same type, but are more irregular and 'embryonic' in form (*Fig. 468*). They are spindle and spheroidal (*Fig. 461*), the cell boundaries are poorly defined, the nuclei very active, and numerous mitotic figures are present (*Fig. 462*). A variable amount of intercellular material is present. The spaces observed in the other growths are present here also. There is much more variation in their size and form, and the surface is much less regularly delimited (*Fig. 465*).

The lining cells may be multi-layered. Papillary projections occur (*Figs. 466, 473*), and these resemble those seen in the other form of growth except that the cells are more anaplastic (*Fig. 467*). Examining any one portion carefully, a marked resemblance to the tumours of synovial membrane, and even to hyperplastic synovial membrane itself, is to be observed. (Cf. *Figs. 464, 460, and 469*.)

The vessels are similar to those common in other malignant connective-tissue neoplasms. They are vascular spaces lined usually by the cells of the tumour, and vary in size and shape. I consider it probable that some of the angiomatous tumours of the tendons are true synovial growths which are somewhat vascular, and in which the gross similarity of the synovial spaces to the vascular spaces has led to the opinion that all are vascular in origin. (*See Fig. 463*.)

It is necessary now to consider in detail the structure of synovial membranes.



FIG. 447.—Photomicrograph of a portion of a tumour. The cells are of the spheroidal type and some degeneration is present. (See Fig. 459.) ($\times 25$.)



FIG. 448.—Another portion of the tumour shown in Fig. 447. The mucoid material, which macroscopically closely resembled that seen in a ganglion, is shown. ($\times 25$.)

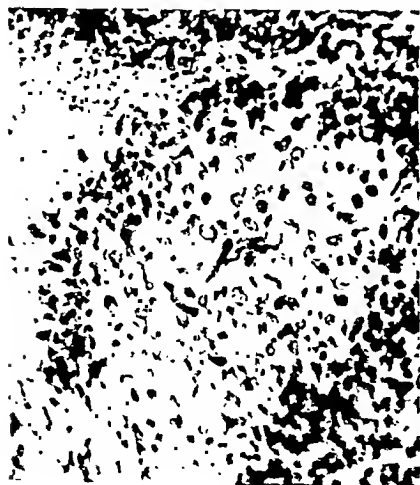


FIG. 449.—A higher-power view of the tissue seen in Figs. 447, 448. The irregularity in the size of the nuclei is seen. (See Fig. 454.) ($\times 160$.)



FIG. 450.—Section of a tendon-sheath tumour showing a number of 'synovial' spaces. ($\times 80$.)

NORMAL HISTOLOGY OF SYNOVIAL MEMBRANES.

A discussion of the histology of the synovial membranes resolves itself into several subdivisions: (1) The synovial cells; (2) The varieties of structure of membranes; (3) Villus formation; (4) Transitions of structure to cartilage formation; (5) Vascular channels.

1. The Synovial Cells.—As previously stated, the 'synovial membrane' consists of a specialized free connective-tissue surface (*Fig. 471*). The cells are of a modified connective-tissue type: some are arranged along the surface, and may be very numerous in some areas (*Fig. 472*), giving the 'cell-rich' type of membrane described by Hammar. In other parts the cells are fewer, giving the 'cell-poor' type (*Fig. 469*). This layer merges gradually into the subjacent tissue, so that no line of demarcation can be found. The cells are surrounded by collagenous tissue. Some of them are on the surface; others are on the surface only in part, or send processes to the surface, while others are completely embedded. Spheroidal cells are found in the cellular areas.

The cell body is usually very irregular in shape, and possesses several small blunted processes. The protoplasm is pale, homogeneous, and stains faintly with eosin. It contains a small number of mitochondria and there is extremely little intracellular fat. Occasionally vacuoles are present in the cells, but the nature of the contents of these spaces is unknown (Key). The nuclei stain deeply and contain a coarse chromatin network. Various forms have been described by Hammar (branched, triangular, curved, spiral, and pyknotic nuclei). These indicate, however, no essential difference, but are merely due to mechanical, physiological, or age alterations. Sometimes the synovial cells may have more than one nucleus.

2. Variations in the Structure of Synovial Membrane.—Key has pointed out that the actual synovial cells are of the same form in all parts of joints and bursæ, etc., but that the underlying tissue is different in different positions. Thus he describes an areolar type, a fibrous type, and an adipose type of synovial membrane. It is not necessary to discuss these varieties here, except to mention that the peculiarities of the synovial cells in relation to the underlying fibrous or fat tissue is mimicked closely by some of the neoplasms. In this respect the adipose type is of special interest. The synovial cells cover the surface of the fat tissue and a few cells may lie between the fat cells. The synovial cells are surrounded by collagenous material and they may be cuboidal in form. In some of the tumours where there are a large number of foamy cells, these fat-containing cells may bear the same relationship to the cells of the tumour as do the ordinary fat cells in adipose synovial membrane (*see Fig. 457*).

3. Villi.—These are circumscribed projections of the synovial surface of a joint or bursa. They occur in cases of chronic inflammation of tendon-sheaths and in tumours, and are bordered by the synovial cells in the same manner as other parts of the 'membrane'. Usually they are much more cellular than other portions and are very vascular. They correspond in structure with the portion of tissue from which they arise, and may be areolar, fibrous, or, to a less extent, even fatty in type.



FIG. 451.—Photomicrograph showing the appearance presented by papillary projections into a space occurring in a tendon-sheath tumour. Compare with higher-power view (Fig. 460), and with inflamed joint synovial membrane (Fig. 472). Cells of inflammation are present in the papillae. ($\times 80$.)

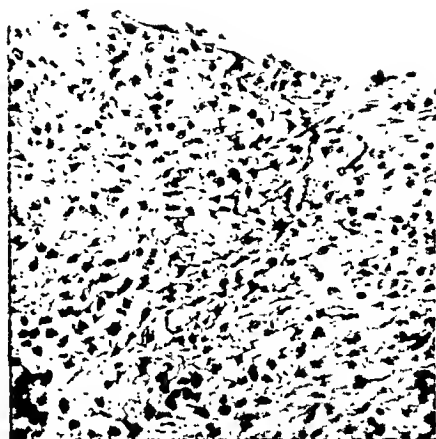


FIG. 452.—Photomicrograph showing the tumour tissue lining one of the spaces shown in Fig. 442.

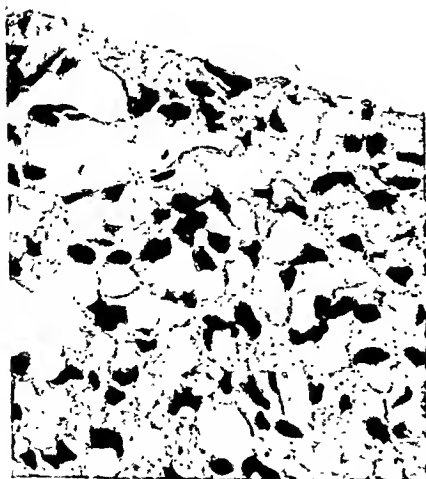


FIG. 453.—High-power view of the tissue shown in Fig. 452. Compare with the synovial membrane shown in Figs. 469 and 470. ($\times 540$.)



FIG. 454.—High-power view of the tissue shown in Figs. 447-449. ($\times 720$.)

4. **Transition of Synovial Cells to Other Forms.**—In the fibrous areas the cells are arranged parallel with each other. In some portions, particularly near the junction of the membrane with cartilage, the cells are more widely separated and irregularly spaced. They lose their processes and they become enclosed in lacunæ, and a gradual transition from the typical synovial tissue through a pseudo-cartilage to a true cartilage occurs. In consideration of this normal relationship of the synovial and cartilage cells, the occurrence of cartilage in 'synovial membrane' is readily understood. Bone also occurs at times. These relationships are very important in the consideration of the so-called 'chondroma' of tendon-sheaths.

5. **Blood- and Lymph-vessels.**—Synovial tissue is richly supplied with both blood and lymphatic vessels. The relationships of these last have not been completely elucidated. A direct connection of the lymphatic vessels with synovial cavities is now held to be improbable. Many blood-vessels are present, particularly in the villi. It is apparent that the endothelial cells lining these vessels morphologically resemble the synovial cells, so that vascular spaces can be interpreted only after careful examination.

DISCUSSION.

Tumours of tendon-sheaths possess features which have attracted considerable attention and led to many differences of opinion. The first histological investigations led to the conclusion that the tumours were sarcomata, e.g., sarcoma globo-cellulaire, sarcoma fuso-cellulaire, myxofibrosarcoma, etc. Later, other interpretations were made—for example, Bellamy regarded the tumours as being endotheliomata. In 1913 Fleissig suggested that the giant-cell tumours were not neoplastic but chronic inflammatory conditions, and a similar opinion was expressed by Dunn. This was another distinct advance in our appreciation of these growths.

The interesting feature of most of the investigations into these tumours is that they refer almost entirely to the presence of the giant cells, foamy cells, etc., and these are discussed to the almost complete exclusion of any other morphological characteristics. Giant cells occur in many circumstances, as do also the foamy cells of xanthomata, so that it is to the type cell of the tumours that we must look for an understanding of these growths. Since the tumours arise from the sheaths of the tendons, a knowledge of their normal histology is essential. Not much is known concerning their structure except that it is similar to the lining of joints and bursæ. This lining is a specialized connective tissue (*Fig. 470*), and the characters of the cells are duplicated in the cells of the tumours derived from the synovial membrane (*Fig. 460*)—in this case the synovial lining of the sheath of the tendons.

The specialized cells of the synovial membrane form a surface for the tissue surrounding the synovial space. The cells of the tumour, in a similar manner, form the lining of spaces which occur throughout the growth (*Figs. 443, 452, 456, 457, 459, 465, 466*). This feature is one of the characteristics of this form of tumour, and has been overlooked in the too close attention devoted to other features. These spaces appeared to have caused Bellamy to regard the growths as being endotheliomata, but he failed to appreciate



FIG. 455.—Section of a tendon-sheath tumour showing the tissue merging into a cartilage-like structure. ($\times 80$.)



FIG. 456.—Photomicrograph of a portion of the tumour seen in Fig. 443. The cells contain a large amount of fatty material, but the characteristic spaces are present. ($\times 80$.)



FIG. 457.—High-power view of the tissue shown in Fig. 456. The 'synovial' cells are observed lining the space and also lying between fat-laden cells. ($\times 540$.)



FIG. 458.—Section showing papillae occurring in a tumour. There is a resemblance to the appearances observed in a chronic tenosynovitis. ($\times 80$.)

that the spaces are not lined by endothelial cells. A casual glance at some sections gives the impression that an endothelial lining is present, but a more complete examination shows that the morphology and arrangement of the cells differ from those of endothelium.

The type cells of the tumours have been discussed. They are spindle and spheroidal in shape, and these forms merge from one into the other and apparently proliferate, with the formation of spaces in the tissue which are comparable with synovial cavities. They are similar to those seen in synovial membranes. The occurrence of synovial spaces amongst the cells should be emphasized. These have been mistaken for vascular channels, blood or lymphatic, so that the evidence for the view that they represent synovial spaces must be considered. These cavities are large elongate hiatuses lined by tumour cells. They are usually much larger than blood or lymphatic spaces in tumours of this character, especially in the more slowly growing innocent varieties. Also they are different from the vascular spaces which are present in the tumours.

The cells lining the spaces are the cells of the tumour; endothelial cells are absent, and the same arrangement of cells as is present in the normal synovial membrane occurs. Immediately subjacent to the surface there may be fibrous tissue or fatty tissue (in the case of the 'xanthomatous' tumours, lipoid-containing cells), as occurs in the normal synovial membranes. Blood-corpuscles are usually absent, and in the cases in which I have observed them they have been present extravasated also throughout the tumour, suggesting that their presence was, in all probability, due to the trauma of operation.

Papillary projections occur in some of these spaces (*Figs. 451 and 458*), particularly in the malignant forms (*Fig. 466*), and though intravascular papillary developments have been described in endotheliomata, those observed in the tendon-sheath tumours resemble more closely the papillary projections observed in the cases of chronic inflammation of joints or in chronic tenosynovitis (Forgue and Étienne).

The mechanism of formation of the synovial spaces is of interest. In the more slowly-growing varieties, a simple split appears to occur in the direction of the parallel intercellular fibrils and cells (*Fig. 443*). In the more rapidly growing varieties, however, a condensation or accumulation of cells occurs along a definite line (*see Fig. 475*). This is reminiscent of the 'condensation' of cells which occurs in embryological tissues prior to the formation of a lumen. Later, a line of cleavage occurs in the region of the proliferated cells (*Fig. 476*). Whether this occurs by degeneration of, or secretion from, the cells is at present undetermined, but the appearances suggest to me that it occurs by secretion. It is to be appreciated that this interpretation of the formation of lumina is purely hypothetical. The great difficulty in drawing deductions in pathological histology is that it is necessary to "translate spatial relationships into those of time". This is very likely to lead one into error. With this consideration in mind, however, I feel that the evidence, though incomplete, is strongly in favour of the suggestion put forward above.

In the most malignant tumours it is in the cellular areas that the 'synovial' spaces occur, so that it is probable that the method of formation is similar; but in these cases the mechanism is more difficult to follow and

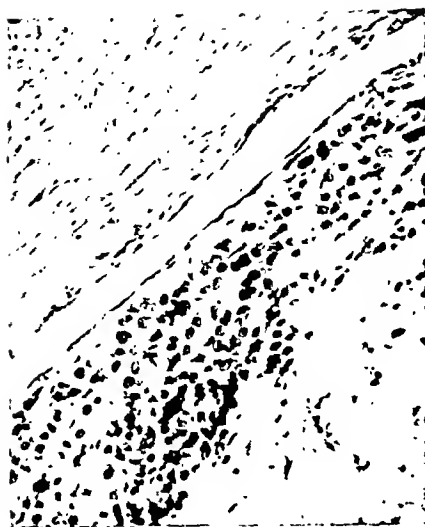


FIG. 459.—A portion of the tumour shown in Fig. 447. A typical space is present, having spindle cells on one aspect and spicoid cells on the other. Mucoid material is present amongst these round cells. ($\times 80$.)

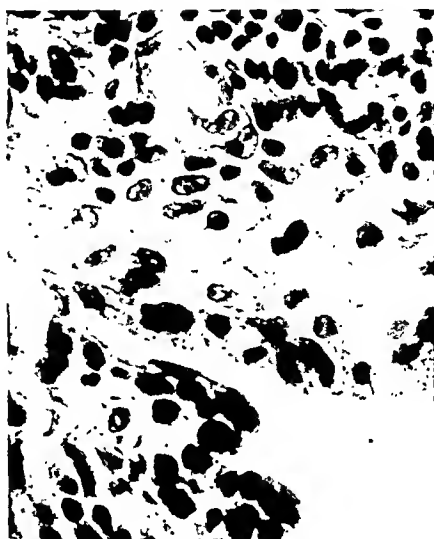


FIG. 460.—Photomicrograph showing the cells lining a space which occurred in one of the tumours. (See Fig. 451.) ($\times 540$.)

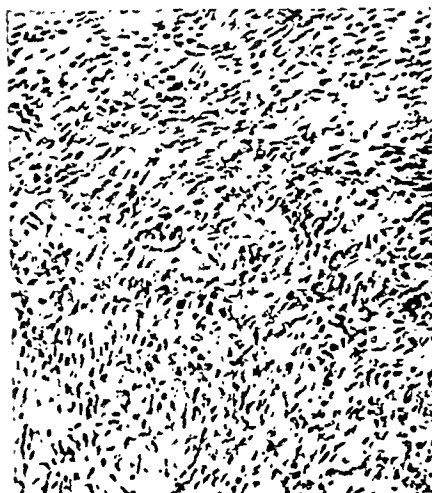


FIG. 461.—Photomicrograph of a portion of a malignant tendon-sheath tumour. This appearance would be labelled spindle-cell or mixed-cell sarcoma. (See Fig. 466.) ($\times 70$.)



FIG. 462.—Higher-power view of the tumour shown in Fig. 461. ($\times 450$.)

interpretation is even more liable to error (*Fig. 473*). The papillary growths arise from the excess focal proliferation of the cells in one area. This forms a papillary projection from which secondary papillae may arise. These are usually very vascular. Often the cells on the surface resemble endothelial cells, but close investigation shows that this is only a superficial similarity. The structure of the protoplasm suggests that they have a secretory function, but this is not proven at present. Droplets similar to those seen in these cells have been observed in the cells of the normal synovial membrane, but it has not been possible to stain them (*Key*).

Another interesting discovery in these tumours is that of tissue resembling cartilage (*Fig. 455*), and even, in some cases, actual cartilage. Examination of synovial membrane in the region of joints shows that the synovial cells merge, by insensible gradations, into the cartilage cells of the articular surfaces of the bones—that is, the synovial cells in certain situations, and presumably under certain influences (biophysical or biochemical), may give rise to cartilage. If the cells, therefore, possess the potentialities for the production of cartilage as well as other varieties of connective tissue, it is unnecessary to postulate cell-rests of primitive osseous or cartilaginous tissues in the sheath of the tendon at the site of the growth in order to explain these aberrant tissues. This occurrence is merely the expression of the capabilities of the cells towards the formation of other tissues when exposed to the adequate stimulus, and applies also to the production of mucoid tissue and bone.

This brings us to the problem of terminology and classification, which has been a somewhat vexed question. The nomenclature includes giant-cell tumour, myeloma, myeloid sarcoma, giant-cell sarcoma, myeloid tumour, xanthoma, myeloxanthoma, myeloid endothelioma, granuloma, and round- and spindle-cell sarcoma, amongst other names. In 1923 Buxton attempted this classification: (1) Fibroma, chondroma, ganglion; (2) Sarcoma. There are several disadvantages in such a classification. The term 'fibroma' is applied to a definite type of growth of the fibrous connective variety, and though the tumour here referred to consists of fibroblasts, many of these are specialized and give the neoplasm a distinctive microscopical picture. Similarly the term 'chondroma' in itself gives but an inadequate conception of the growth. Little need be said concerning the term 'sarcoma'. The sarcomata have in recent years gradually been reduced from a numerically impregnable position, by the segregation of the 'sarcomata' of the testis, retroperitoneal tissues, mediastinum, etc., until now, with the exception of the osteogenic sarcomata, they comprise a decimated band which is viewed with considerable suspicion. No diagnosis is satisfactory, therefore, unless it is sufficiently complete to indicate the histogenesis and nature of a malignant connective-tissue neoplasm. Such terms as 'round-cell sarcoma' and 'giant-cell sarcoma' are not only futile, but are also treacherous, since they cause us to pigeon-hole a tumour whose origin is undetermined.

Since the type cell is distinctive, the term 'teno-synovioma' completely describes the growth. Multiplicity of terminology is to be deprecated, but in some departments of pathology we are without adequate or sufficient terms. Probably the term 'synovioma' is sufficient, since the growths arising



FIG. 463.—Another portion of the tumour shown in *Figs. 461, 462*. Such growths have been described as endotheliomata. ($\times 80$.)



FIG. 464.—Higher-power view of cells lining the spaces in *Fig. 463*. Compare with cells lining the joint cavity in *Fig. 472*. ($\times 540$.)

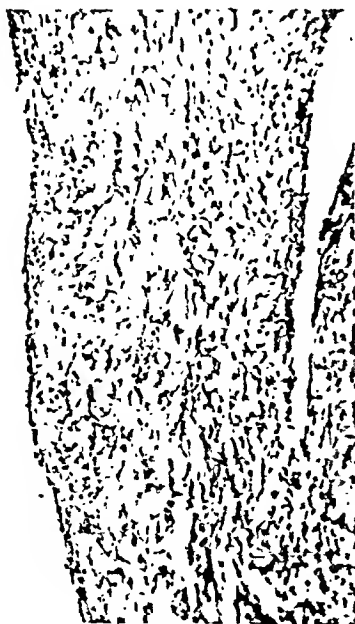


FIG. 465.—Photomicrograph of a section taken from a recurrent tumour. ($\times 80$.)



FIG. 466.—Section showing an irregular space with atypical papillary projections in a malignant tendon-sheath growth. ($\times 80$.)

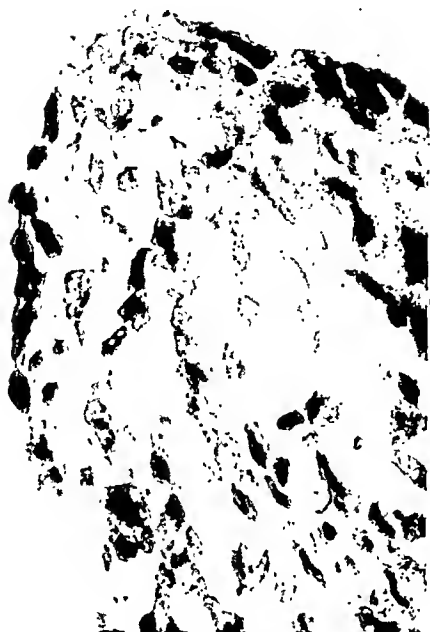


FIG. 467.—Higher-power view of the cells lining one of the projections shown in *Fig. 466*. ($\times 540$.)



FIG. 468.—Photomicrograph showing the cells comprising the growth shown in *Fig. 466*. Compare them with those of the inflamed synovial membrane in *Fig. 470*. ($\times 540$.)



FIG. 469.—Photomicrograph of a portion of synovial membrane, showing the cells lying either on the surface or embedded more or less completely in the connective tissue. ($\times 540$.)

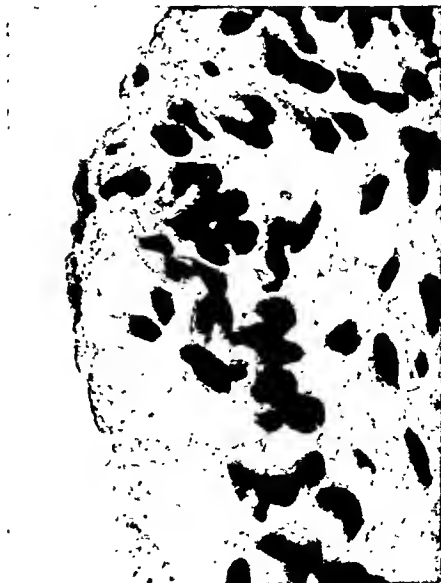


FIG. 470.—Section of chronically inflamed synovial membrane. The cells are spindle or spheroidal in shape, and do not line the surface as do endothelial cells. ($\times 540$.)

in the synovial membrane of joints present certain similarities to these discussed. As I am, at the moment, without sufficient material to demonstrate this, the first term is here retained. It should be appreciated that this is not a suggestion that this term should be brought into general use, but is merely an attempt to bring certain of the special features of these growths into general notice. The classification suggested is:—

TENO-SYNOVIOMA—

Innocent:

Fibrous

Cartilaginous

Fatty

Vascular

Ossous

Mixed forms containing several of these tissues.

These may be complicated by the presence
of giant cells or foamy cells.

Malignant.

The giant cells and the foam cells require special mention. As previously stated, these cells have absorbed the attention of many investigators to the exclusion of all else. The giant cells are of the foreign-body variety. They have been shown to be associated in these growths with the presence of free cholesterol. Elsewhere, the association of giant cells with cell debris, material difficult of absorption, fatty material, etc., has been demonstrated frequently. There is no reason for considering that they have greater significance in this case than the absorption of degenerated material.

In any attempt to determine the histogenesis of these neoplasms it is imperative that we ignore these features which are incidental in the life-history of the growth. This is the more necessary since, previously, so much emphasis has been laid on their presence. In a similar manner, the foam cells must be regarded as incidental. They occur in many parts of the body where there has been injury or hæmorrhage, and the foam cells of tendon-sheath tumours are probably also such phagocytic cells. These foam or 'xanthoma' cells are the cause of the yellow colour observed in some of these growths. A band of yellow colour running through a tumour will be found to correspond microscopically to a band of tissue which consists of these cells.

The relationship of the tendon-sheath tumours to ganglion is another problem requiring elucidation. As shown in the description of the macroscopic and microscopic appearances, areas almost identical, as far as could be determined, with a ganglion were found in one of the tumours examined. The similarity extended even to the presence of millet-seed bodies. It would appear that a ganglion may arise as a tumour which has undergone mucoid change.

Is the condition inflammatory or neoplastic? This is a question that has been asked many times. There are a certain number which are undoubtedly neoplastic, but some—particularly the giant-cell forms—are more difficult of interpretation. The xanthoma cells are very common in inflammatory conditions, and are unknown as composing a neoplasm. Giant cells also, of the foreign-body type, are evidence of tissue absorption—and hence destruction



FIG. 471.—Photomicrograph of a section showing synovial fringes from a case of osteoarthritis. The character of the lining tissue is well shown. ($\times 80$.)



FIG. 472.—Photomicrograph of a portion of an inflamed synovial membrane. The cells are spheroidal in shape and do not lie on the surface. ($\times 540$.)



FIG. 473.—Photomicrograph of a portion of a malignant tendon-sheath tumour. ($\times 80$.)

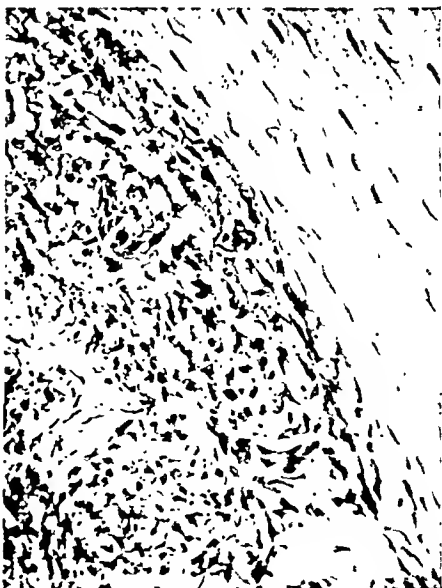


FIG. 474.—Photomicrograph of a portion of the growth shown in *Fig. 442*, showing the invasion of the tendon by the neoplasm. ($\times 160$.)

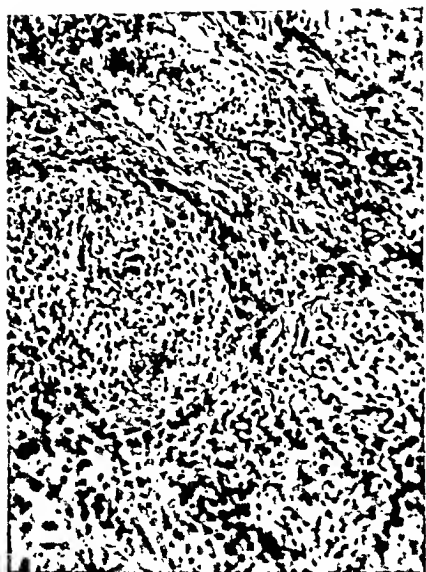


FIG. 475.—Photomicrograph of a portion of a tendon-sheath tumour showing the curved line of 'condensation' of the cells, which is thought to occur prior to the formation of a cavity. (Cf. with Fig. 476.) ($\times 80$.)



FIG. 476.—Another portion of tissue from the same tumour as shown in Fig. 475, showing the formation of a 'synovial' space. ($\times 160$.)

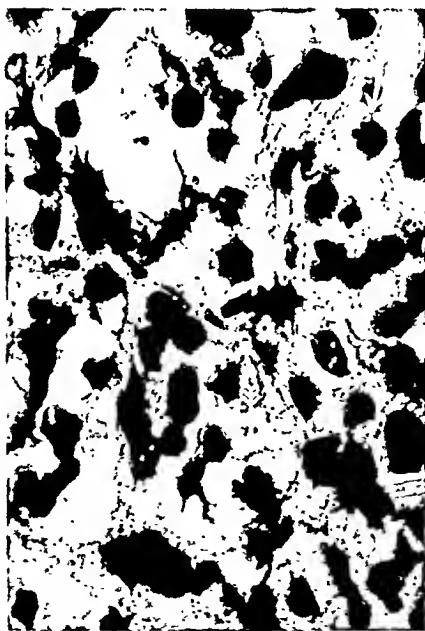


FIG. 477.—Photomicrograph of a portion of a tumour which closely resembled a ganglion. Note the number of mitotic figures—indicated by arrows. ($\times 720$.)

—and usually indicate an inflammatory process. The history of trauma in most cases suggests that the destruction of tissue may give rise to subsequent inflammatory reaction and growth. On the other hand, true new growths may arise after an injury, and, as stated, some of the tumours under discussion are undoubtedly neoplastic. Some of the tumours that I have examined, though showing evidences of chronic inflammation, possess so many mitotic figures in the cellular areas (*see Fig. 477*) that a neoplastic element seems certain. We may conclude, therefore, that some of the tendon-sheath tumours are certainly neoplastic, and others are lowly malignant or innocent growths with a superadded chronic inflammation.

The definite inflammatory conditions—e.g., chronic tenosynovitis—are beyond the scope of this paper. Dunn has suggested that the doubtful tumours are on the borderline between inflammation and new growth. Since there appears to be every gradation between the malignant metastasizing tumour through the innocent growth to the chronic inflammatory conditions, some will no doubt fall into this class. A brief consideration of the definition of neoplasia will prevent our being dogmatic on this subject. Most statements in our present state of knowledge must be a question of opinion.

SUMMARY.

1. Synovial membranes have a characteristic histological picture.
2. Tendon-sheath tumours may be interpreted in terms of this normal cellular structure.
3. 'Synovial spaces' in tumour tissue, and not the giant cells or xanthoma cells, are an important and characteristic feature.
4. Metaplasia, the formation of cartilage, and the occurrence of chondroma are discussed.
5. The relationship to ganglion is mentioned.
6. The views concerning the neoplastic and inflammatory origin of some of the growths are reviewed.

CONCLUSIONS.

Pathological conditions are to be explained in terms of the characteristics of the tissues in which they arise.

Tumours of tendon-sheaths arise from the cells of the synovial membrane of the sheath, and the morphology and nature of the growths depend on the potentialities of the originating cells.

Classifications of these growths should embody this principle.

BIBLIOGRAPHY.

Histology of Synovial Membranes.—

- BENTZEN, G. E., *Nord. med. Arch.*, 1875, vii, 1.
 BORCHARDT, M., *Arch. f. klin. Chir.*, 1900, lxii, 443.
 BRAUN, H., *Deut. Zeits. f. Chir.*, 1894, xxxix, 35.
 COLOMIOTTI, J. V., *Giorn. d. R. Accad. d. Med. d. Torino*, 1876; quoted by Key.

- HAGEN-TORN, O., *Arch. f. micro. Anat.*, 1882, xxi, 589.
 HAMMAR, A., *Ibid.*, 1894, xliii, 266, 813.
 HEINE, J., *Virchow's Arch.*, 1926, cclx, 521.
 HUETER, C., *Arch. f. pathol. Anat.*, 1866, xxxvi, 25; *Dent. Zeits. f. Chir.*, 1876, vi, 290.
 KEY, J. A., *Jour. Bone and Joint Surg.*, 1925, vii, 793; *Special Cytology*, 1928, New York.
 LANDZERT, T., *Zentralb. f. med. Wissens.*, 1867, xxiv, 370.
 LUBSCH, W., *Bau und Entstehung der Wirbeltiereelenke*, 1910, Zena.
 REYHER, C., *Jour. Anat. and Physiol.*, 1874, viii, 261.
 SCHWEIGGER-SEIDEL, F., *Arbeit. aus d. physiol. Anstalt z. Leipzig*, 1867, i, 150.
 SEGAL, C., *Beitr. z. klin. Chir.*, 1913, lxxxvii, 259.
 SNEEDS, R. T., *Contrib. Embryol. Carnegie Institute*, 1923, xv, 55.
 VAN DER SLUYS, *Nederl. Arch. f. Zool.*, 1876, iii, 83; quoted by Key.
 SOUBBOTINE, M., *Arch. de Physiol.*, 1880, vii, 532.
 STEINBERG, "Researches on the Structure of Synovial Membrane", Inaug. Diss. (St. Petersburg): quoted by Soubbotine.
 SUMITA, M., *Arch. f. klin. Chir.*, 1912, xcix, 755.
 TILLMANS, H., *Arch. f. micro. Anat.*, 1874, x, 401; *Arch. f. klin. Chir.*, 1875, xix, 693.
 TOURNEUX, F., and HERRMAN, G., *Gaz. méd. de Paris*, 1880, xix, 149.
 WEICHELBAUM, *Die seiten Veränderungen der Gelenke und deren Zusammenhang mit der Arthritis*.

Tumours of Tendon-sheaths.—

- AGRIROGLIO, M., *Ann. ital. di Chir.*, 1920, vii, Nov. 30, 1220.
 ALARY, "Contribution à l'Étude des Tumeurs sarcomateuses des Gaines tendineuses", *Thèse de Toulouse*, 1912.
 ALBERTIN and PAVIOT, "Sarcome à myéloplaxes des Fléchisseurs de la Main", *Province méd.*, 1899.
 ARCOLEO, E., *Riforma Med.*, 1899, xv, 315.
 BAZY, P., *Bull. Soc. anat. de Paris*, 1907, 6^{me} s., ix, 472.
 BELLAMY, H. F., *Jour. Pathol. and Bacteriol.*, 1901, vii, 465.
 BERTI, G., *Tumori*, 1923-4, x, 469.
 BILLROTH, "Schnenscheidensarkome", *Chir. Klin. in Wien*, 1868-9, quoted by Tourneux.
 BLANC, "Sarcome primitif des Gaines tendineuses", Société des Sciences médicales de St. Etienne, *Loire méd.*, 1912, 118; quoted by Tourneux.
 BOECKEL, A., *Province méd.*, 1913, 36.
 BOLOGNESI, "Étude sur les Tumeurs des Gaines synoviales du Poignet", *Thèse de Paris*, 1882.
 BONHOMME, P., "Contribution à l'Étude des Tumeurs myéloïdes des Gaines tendineuses", *Thèse de Lyon*, 1897.
 BONJOUR, S., "Contribution à l'Étude des Tumeurs fibro-tendineuses à Myéloplaxes", *Thèse de Paris*, 1897.
 BRANDAO, A., *Chir. d. Org. di Mov.*, 1925, ix, 235.
 BRECKMANN, E., and JUNG, A., *Rev. d'Orthop.*, 1929, xvi, 176.
 BROCA, P., *Bull. et Mém. Soc. de Chir.*, 1860, 2^{me} s., i, 342.
 BRODERS, A. C., *Ann. of Surg.*, 1919, lxx, 574.
 BUNTON, ST. J. D., *Brit. Jour. Surg.*, 1923, x, 469.
 CASSAËT and LABOUGLE, J., *Jour. de Méd. de Bordeaux*, 1890, xix, 271.
 CHASSAIGNAC, *Gaz. des Hôp. civils et militaires*, 1852, 185.
 CHAUVAIN-ROUX, *Bull. et Mém. Soc. anat. de Paris*, 1920, xe, 192.
 COENEN, H., *Arch. f. klin. Chir.*, 1906, lxxviii, 711.
 CZERNY, V., *Ibid.*, 1869, x, 904.
 DEGORCE, A., *Bull. et Mém. Soc. anat. de Paris*, 1901, lxxvi, 557.
 DENCÉ, *Ibid.*, 1885, 4^{me} s., x, 109.
 DOR, L., *Cong. franç. de Chir.*, 1898, session xii, 553; *Rev. de Chir.*, 1898, xviii, 1089.
 DUMBATH, "Zur Kenntnis der Schnenscheidensarkome", Inaug. Diss., Würzburg, 1886.
 DUNN, J. S., *Trans. Med.-Chir. Soc. Glasgow*, 1914, xiii, 110.
 DURANTE, L., *Chir. d. Org. di Mov.*, 1923, vii, 392.
 EICHURST, "Stat über die von Bändern und Gelenken ausgehenden Geschwulste", Inaug. Diss., Halle, 1876.
 ELY, L. W., *Ann. of Surg.*, 1918, lxviii, 426.
 FALDINI, G., *Chir. d. Org. di Mov.*, 1928, xii, 417.
 FERRÉ, *Bull. et Mém. Soc. anat. de Paris*, 1888, 5^{me} s., ii, lxiii, 312.
 LE FILLIATRE, *Ibid.*, 1913, lxxviii, 222.
 FLEISSIG, J., *Dent. Zeits. f. Chir.*, 1913, cxvii, 239.

- FRITSCH, K., *Beitr. z. klin. Chir.*, 1908, lx, 344.
- GAUDIANI, V., *Policlinico (Sez. Chir.)*, 1906, xiii, 547; 1908, xv, 272.
- GIGNOUX, "Contribution à l'Étude des Sarcomes des Gainés tendineuses", *Thèse de Montpellier*, 1911.
- GRANT, T. P., and STEWART, M. J., *Glasgow Med. Jour.*, 1914, lxxxi, 333.
- GROSS, *Bull. et Mém. Soc. de Chir.*, 1878, iv, 284.
- HARBITZ, F., *Arch. of Pathol. and Lab. Med.*, 1927, iv, 507.
- HARTERT, W., *Beitr. z. klin. Chir.*, 1913, lxxxiv, 546.
- HELLER, "Zur Kenntniss der Fibrome und Sarkome an Hand und Fingern", *Inaug. Diss.*, Leipzig, 1902.
- HEURTAUX, M. A., *Arch. gén. de Méd.*, 1891, elxvii, 40, 160.
- HOESSLI, H., *Beitr. z. klin. Chir.*, 1914, xc, 168.
- HÜNERMANN, T., *Deut. Zeits. f. Chir.*, 1923, clxxxii, 410.
- JANIK, A., *Ann. of Surg.*, 1927, lxxxv, 897.
- JOURDAN and ÉTIENNE, *Montpellier méd.*, 1911, xxxiii, 372.
- KROGIUS, A., *Acta Chir. Scand.* 1922-3, lv, 363.
- KUSNETZOWSKI, N. J., *Arch. f. klin. Chir.*, 1923, cxxiv, 73.
- LABOUGLE, J., *Jour. de Méd. de Bordeaux*, 1889-90, xix, 177.
- LANDOIS, F., and REID, M., *Beitr. z. klin. Chir.*, 1914, xc, 56.
- LECÈNE, P., and COULONGUET, P., *Ann. d'Anat. pathol.*, 1924, i, 393.
- LECLERC, *Bull. et Mém. Soc. anat. de Paris*, 1879, Nov. 7, 619.
- LENZI, L., and ABETTI, M., *Clin. Chir. Milano*, 1909, xvii, 1617.
- LÉO, G., *Paris Chir.*, 1923, xv, 461.
- LONGUET, L., and LANDEL, G., *Arch. de Méd. expér.*, 1895, vii, 753.
- MALAPERT, P., and MORICHAU-BEAUCHANT, R., *Bull. et Mém. Soc. anat. de Paris*, 1905, lxxx, 390.
- MALHERBE, M., *Rev. de Chir.*, 1896, xvi, 842.
- MANNINI, R., *Policlinico*, 1928, xxxv, 364.
- MARKOE, T. M., *Medical News*, 1884, xlv, 464.
- MARTINI, *Arch. ed Atti d. Soc. ital. di Chir.*, 1902, xliii.
- MAYER, "Ueber maligne Geschwülste der Sehnensehiden", *Inaug. Diss.*, Würzburg, 1886.
- MENCIÈRE, L., *Gaz. hebdom. de Méd. et Chir.*, 1898, No. 7, iii, 73.
- MÉRIEL, E., *Arch. méd. de Toulouse*, 1912, xix, 131.
- MEYER, W., *Ann. of Surg.*, 1897, xxv, 623.
- MONTPROFIT, *Bull. et Mém. Soc. anat. de Paris*, 1891, lxvi, 40.
- MORESTIN, H., *Ibid.*, 1890, lxv, 529.
- MÜLLER, R. F., *Arch. f. klin. Chir.*, 1901, lxiii, 348.
- NOWY, "Contribution à l'Étude des Sarcomes des Gainés tendineuses", *Thèse de Montpellier*, 1909; quoted by Tourneux.
- OLLERENSHAW, R., *Brit. Jour. Surg.*, 1923, x, 466.
- PAQUET, *Bull. et Mém. Soc. de Chir.*, 1878, iv, 705.
- PETZOLD, "Zur Kasuistik der Sehnensehiden Sarkome", *Inaug. Diss.*, Leipzig, 1901.
- DE PEZZER, O., "Tumeurs solides des Gainés synoviales", *Thèse de Paris*, 1880.
- PILLIET, A. H., *Bull. et Mém. Soc. anat. de Paris*, 1893, 5^{me} s., vii, 653.
- PILLIET, A. H., and MAUCLAIRE, *Ibid.*, 1894, viii, 298.
- PINKUS, F., and PICK, L., *Deut. med. Woch.*, 1908, xxxiv, 1426.
- PRINGSHEIM, J., *Ibid.*, 1908, 2145.
- PYBUS, F. C., *Brit. Jour. Surg.*, 1917, v, 172.
- REBOUL, J., *Arch. Prov. de Chir.*, 1892, i, 367, 375.
- REVERDIN, J. L., *Rev. méd. de la Suisse Rom.*, 1885, v, 671.
- ROMITI, Z., *Arch. ital. di Chir.*, 1925, xii, 406.
- ROSENTHAL, A., *Beitr. z. klin. Chir.*, 1909, lxiv, 577.
- RUSSELL, J. I., *Ann. of Surg.*, 1911, liii, 285.
- SEGOVIA, J., and LLOMBART, A., *Arch. de Med. Cir. y Espee.*, 1928, xxviii, 10.
- SENDLER, P., *Zentralb. f. Chir.*, 1891, xviii, 537.
- SCHULZ, R., *Virchow's Arch.*, 1884, xc, 123.
- SCHWARZ, *Münch. med. Woch.*, 1908, lv, 1235.
- SOUBEYRAN, "Fibro-myxo-sarcome des Gainés des Extenseurs du Pied", *Thèse Koupper, Montpellier*, 1910; quoted by Tourneux.
- SPENCER WELLS, T., *Trans. Pathol. Soc. Lond.*, 1857, 379.
- SPIESS, P., *Frankf. Zeits. f. Pathol.*, 1913, xiii, 1.
- SPRENGEL, *Centralb. f. Chir.*, 1888, xv, 153.
- STEWART, M. J., and FLINT, E. R., *Brit. Jour. Surg.*, 1915, iii, 90.
- STRAUSS, A., *Surg. Gynecol. and Obst.*, 1922, xxxv, 161.
- SULZER, "Ueber Geschwülste an des Gelenkenden", *Inaug. Diss.*, Halle, 1869.

- TARGETT, J. H., *Trans. Pathol. Soc. Lond.*, 1897, xlviii, 230.
 THORN, J., *Arch. f. klin. Chir.*, 1896, lii, 593.
 TICHOFF, P., "Lipoma Arborescens": quoted by Strauss.
 TOMASELLI, G., *Riforma Med.*, 1901, xviii, 770.
 TOREK, F., *N.Y. Med. Jour.*, 1905, lxxxii, 20.
 TOURNEUX, J. P., *Rev. de Chir.*, 1913, xlvii, 817; *Progrès méd.*, 1920, xxxv, 215.
 VENOT, A., *Rev. de Chir.*, 1898, xviii, 232.
 WEIL, S., *Beitr. z. klin. Chir.*, 1914, xciii, 617.
 WHITE, J. R., *Surg. Gynecol. and Obst.*, 1924, xxxviii, 489.
 ZWICKE, "Zur Kenntnis der Sehnen Scheidensarkome", *Charité Ann.*, 1883.

THE TREATMENT OF CEREBRAL TUMOURS WITH RADIUM.*

BY J. PATERSON ROSS, LONDON.

(Being a Hunterian Lecture delivered at the Royal College of Surgeons of England on January 28, 1931.)

THE treatment of intracranial tumours by any method at present conceived presents certain problems peculiar to this field of surgery. There are difficulties of diagnosis, both anatomical and pathological; difficulties of access; and complications which arise from sudden changes in tension within the dural capsule, with resultant alterations in the circulation and damage to the cerebral tissue. Gradually these problems have been defined and tackled, and modifications in technique have been introduced to improve the methods of approach to the brain, to obviate dangerous changes in intracranial tension, to reduce the risk of infection to the minimum, and to control hæmorrhage. The recent adaptation of electrosurgical methods, the exact value of which is at present difficult to estimate, has been acclaimed the most notable advance in surgery since the introduction of antiseptics.

Technical advances have led to fresh discoveries in regard to the pathology of brain tumours, and have rendered possible and successful procedures which before the beginning of the present century would have proved too hazardous. This applies especially to the treatment of encapsulated tumours, but in the case of the gliomas it must be confessed that attempted removal, although carrying a much lower immediate mortality-rate than formerly, gives but a small chance of ultimate cure. Seeing that, if pituitary tumours be excluded, the glioma group constitutes over 70 per cent of the primary brain tumours verified by operation, the outlook is rather discouraging, and the hope has been expressed that radiotherapy might succeed where surgery has failed. The present studies have been undertaken to try to discover how far this hope is justifiable. The investigation is confined to the glioma group, though there are many recorded observations in regard to irradiation of endotheliomas, pituitary tumours, and neurofibromas.

PATHOLOGICAL CONSIDERATIONS.

In order to understand the problem of the gliomas it is necessary to make a brief reference to their pathology. Of the many criteria which indicate the malignancy of a tumour, the most important is the character of the neoplastic cells, and especially the degree to which they are differentiated. The more embryonic and undifferentiated the cells, the more active is the growth and the more malignant the tumour; and there is evidence that the

* From the Surgical Professorial Unit, St. Bartholomew's Hospital.

degree of differentiation may vary during the life of a glioma, the tendency being towards de-differentiation (Bailey). Guided by this histological feature, it is possible to grade the gliomas according to their malignancy, although they all infiltrate the brain and none disseminates by the lymphatic or blood-stream.

At an early stage in the development of the nervous system from the embryonic epiblast two main types of cell are distinguishable—the primitive spongioblast and the germ-cell. From some of the primitive spongioblasts the ependyma is derived, and the remainder eventually become the astrocytes of the adult neuroglia. The astrocyte has multiple processes which can be demonstrated by gold staining methods, and one of these processes is thicker and longer than the others and is attached to the wall of a blood-vessel, where it is flattened out into a small end-plate known as a 'sucker foot'. The supporting tissue of the nervous system may be regarded as a network formed of the processes of the astrocytes stretching across from vessel to vessel. The germ-cells give rise to neuroblasts which develop into nerve-cells, and to medulloblasts.

The gliomas have been separated by Bailey and Cushing¹ into thirteen histological groups, but in their large series they found that between 35 and 40 per cent were astrocytomas, 35 per cent spongioblastomas, and 13 per cent medulloblastomas, these three groups thus accounting for about 85 per cent of the verified gliomas. Carmichael,² analysing a smaller series, also found that almost all the gliomas belonged to these three groups, but that the proportion of spongioblastomas to astrocytomas was considerably greater.

Astrocytomas are composed of cells which resemble closely the adult astrocytes, having an affinity for gold stains, and each cell possessing one long process attached to a vessel by a 'sucker foot'. The tumour appears as a white, firm area, and, having no recognizable edge, it often looks like an enlargement of the white matter of the brain. Not uncommonly the tumour substance becomes liquefied, and a gliomatous cyst is the result. The blood-supply of the tumour is but slightly richer than that of the neighbouring brain, which shows no reactionary gliosis. Growth is slow, and it has been estimated that the average duration of life after signs of tumour formation have appeared is twelve to eighteen months, though it may be considerably longer. There is evidence that certain of the astrocytomas can recur in a more malignant (de-differentiated) form after partial removal.

The spongioblast is a much less differentiated cell than the astrocyte, possessing only one or two short processes which never show a 'sucker foot'. Spongioblastomas are soft tumours, usually reddish in colour owing to their great vascularity and to old and recent hæmorrhages. They seem to have a well-defined margin, and often give a deceptive appearance of encapsulation. Sometimes the majority of the tumour cells may be recognized as spongioblasts, but more commonly the growth consists of a mass of embryonic cells varying greatly in size and shape, and taking gold stains poorly. The peripheral portions of the tumour are cellular, the central degenerate, and what appears macroscopically to be the capsule is seen to be in reality a thin dense layer of growth enveloping a mass of degenerate tissue. The tumour is surrounded by a well-marked zone of gliosis and œdema.

These rapidly growing neoplasms are the so-called gliosarcomas of the older literature (a misnomer due to ignorance of the ectodermal origin of the neuroglia), whose early recurrence after 'enucleation from the capsule' is now recognized as continued proliferation of rapidly growing cells which form the layer mistaken for a capsule. They are usually fatal within about six months of their recognition.

Seeing that these tumours have given rise to so much speculation, it is interesting to try to discover what John Hunter thought about them nearly a hundred and fifty years ago. That he observed them there is no doubt; and the specimen of a cystic spongioblastoma in the Museum of the Royal College of Surgeons (No. 5030-1) is probably the preparation referred to as a hydatid in the brain in lectures delivered by Sir Everard Home "chiefly from Mr. Hunter's notes". The term 'hydatid' signified merely a cyst, and in the lecture notes it is stated that hydatids form in the brain of the human subject. This occurred in a lady in London who had violent and distressing headache, and this increased almost to madness. The pain came on periodically, so that the cause could not be made out, as the symptoms were not like those of depressed brain. She died, and on examining the brain a hydatid was found, of a globular shape and of the size of a common orange.

It would appear that Hunter confused the solid spongioblastomas with chronic abscesses, though he was not satisfied that the appearances accorded with the picture of inflammation. In the *Treatise on Inflammation* he writes: "Chronic inflammation is much more frequently attended with increased vascularity at first, but—as the brain becomes softened—with a red, yellowish, or whitish colour, marking three several kinds of mollescence, depending on the effusion of blood, pus, or serum. There seems reason, however, for believing that this pulpy disorganization of the brain may take place independently of inflammation. The most remarkable circumstance with this tissue is the occurrence of large chronic abscesses, generally enclosed in thick cysts and unattended with any symptoms during life. These I have known to occupy one entire hemisphere of the brain. Ulceration sometimes occurs towards the surface of the convolutions, and by some the diffuent state of the brain before spoken of is thought to resemble mortification."

The medulloblastomas are tumours consisting of still more primitive spheroidal cells without processes. They occur chiefly in children, and always in the cerebellum or its immediate neighbourhood, and their clinical history is very brief. They are very vascular, and owing to the extreme rapidity of their growth they soon break through the covering pia mater and give multiple deposits in the subarachnoid space which have been referred to in the past as 'sarcomatosis of the meninges'.

RESULTS OF SURGICAL TREATMENT.

What has surgery to offer in the treatment of the gliomas? Even in the case of the astrocytomas it is clear that it may be impossible to remove the tumour entirely unless one is prepared to sacrifice some of the surrounding brain tissue as well, and in spite of the controversy over cerebral localization, there are few areas of the brain which we are justified in regarding as

unimportant and which may be removed with impunity. When an astrocytoma is cystic, it is often possible to find, after evacuation of the cyst, a nodule of tumour which can be removed apparently completely. Even in such cases it is impossible to foretell what may happen: some recur as solid, more de-differentiated tumours, but others have been followed up for years and remain apparently cured. The average duration of life after operation on an astrocytoma is about five years. The spongioblastomas and medulloblastomas, however, have characters which render their complete removal practically impossible. By electrosurgery better results may be obtained, but at present the average survival period after operation on a spongioblastoma is under one year, and in the case of a medulloblastoma it is still shorter.

Statistics published from the best-known clinics show that operations upon the gliomas carry a post-operative mortality-rate of between 20 and 25 per cent. Of the patients who recover from the operation, nearly 70 per cent are unlikely to live for a year, and rather less than 25 per cent make a prolonged and good functional recovery. Small wonder that the neurological surgeon should search for a method of improving these results!

POSSIBILITY OF ATTACK BY RADIUM.

The outlook in tumours of other organs has been improved during the past few years by modifications in radium technique, and this prompts one to use similar methods for tumours of the brain. Two questions immediately call for an answer—Is it likely that radium will have any effect on glioma cells? and—Will the dose of radium likely to affect the tumour cells destroy healthy cerebral tissue?

A good deal of study has already been devoted to the effects of X rays upon cerebral gliomas. Many conflicting reports have been published, the growth of some tumours being arrested while others seemed to be unaffected by the treatment. The fullest account has been contributed by Bailey and Sosman³ from Cushing's clinic, and investigations on similar lines from other centres⁴ agree with their conclusions. In all their cases an operation was performed and an attempt made to remove the tumour, the nature of which was thus identified; and their results show the value of X-ray treatment as an adjuvant to operation. They believe that life was prolonged considerably in many patients with medulloblastomas, and in a certain number who had spongioblastomas, though in the latter group hæmorrhage not infrequently took place into the tumour, owing, it is supposed, to the damage done by the rays to the poor blood-vessel walls. The survival period of the astrocytomas was not prolonged at all, and many of the patients seemed to be made worse by the treatment. The general result of these authors' investigation was to confirm Ewing's prediction that the tumours with the more embryonic type of cell would react more favourably to irradiation. Ewing⁵ goes so far as to say that the gliomas present most of the structural features which favour susceptibility to radiation, being cellular tumours whose cells are delicate, whose blood-vessels are numerous and fragile, and which frequently show hæmorrhage and necrosis. In his opinion only the astrocytoma contains anything like a resistant intercellular material, and this is usually

poorly formed and scanty; and he concludes that he knows of no tumour which on *a priori* grounds ought to yield more readily to moderate radiation than the average glioma.

It seems reasonable to suppose that radium should have an effect similar to that of X rays.

EFFECTS OF RADIUM ON NORMAL CEREBRAL TISSUE.

Assuming the use of radium to be reasonable, especially in gliomas composed of cells of the more embryonic types, the more important question of injury to the surrounding brain substance by radium has to be considered. Experimental work on this subject began nearly thirty years ago. The early workers employed large doses of unfiltered rays applied for a short time, using, as a rule, small animals. Hæmorrhage into the irradiated brain substance was the constant result, the severity of the lesion varying as the dose, and inversely as the age of the animal. They concluded that the effect of radium on the brain was to damage the blood-vessels without producing marked change in the nerve-cells, but it must be remembered that these changes may have been due largely to β rays. Such experiments with some modifications have been repeated recently by Bagg,⁶ Pendergrass,⁷ and others, and similar results have been obtained. The greater part of the experimental work recorded is not of direct value to the surgeon, since dosage and filtration are inadequately described, or have been quite out of proportion to what would be used in surgery. Horsley and Finzi⁸ used well-filtered radium, but, as was then the custom, they applied a large dose of radium for a short time—55 mgrm. of radium bromide for two and a half to four hours. Though previous workers had produced paralysis in some of their animals, those operated upon by Horsley and Finzi showed no symptoms, probably because they were excluding almost all the β rays. The animals were killed from two to six weeks later, and the cortex and meninges showed hæmorrhage, thrombosis of vessels, and infiltration of the perivascular spaces. In the thrombotic area the neuroglia was vacuolated and the nerve-cells had disappeared, but any changes in the nerve tissue were believed to be secondary to the vascular lesions. In recent years it has become the practice when treating tumours in other parts of the body to use smaller, more widely distributed doses of radium for longer periods. Bagg had shown experimentally that a small dose of unscreened radon acting for a long time gives a small lesion in the brain, whereas the same dose given as a large quantity of radon acting for a short time caused widespread destruction of cerebral tissue and was often fatal.

E. A. Carmichael and I have carried out a series of experiments to try to discover what the effect would be upon normal brain tissue of well-screened radon and radium applied in doses comparable with those used for the treatment of tumours elsewhere in the body. For the experiments rabbits have been used, and the sources of irradiation have been radon seeds filtered by 0.3 mm. of platinum, radon seeds filtered by 0.5 mm. of platinum, and radium needles containing 0.5 mgrm. of radium filtered by 0.6 mm. of platinum (*Fig. 478*). Under ether anæsthesia an incision is made through the scalp, and the

bone is removed, working forwards from the ridge between the supra-occipital and parietal bones, till an area of dura about 1 by 0.5 cm. has been exposed about 1 cm. from the mid-line. In the early experiments the dura was then incised and the filtered seed placed on the cortex; but in the majority the seed has been placed on the intact dura so as to diminish the risk of wounding the brain. Control experiments with empty platinum containers have been carried out from time to time, and in none has any change been found in the brain. Our first object was to produce a lesion which could be attributed only to irradiation. Therefore doses much larger than the therapeutic dose were used, in the hope that the study of such lesions would indicate what changes should be looked for when therapeutic doses were applied. Twenty-eight experiments have been done with large amounts of radon, the duration of the application and the filtration of the seeds being varied. The average strength of the seed at the time of insertion has been 5 mc., and in sixteen cases the seed was filtered by 0.3 mm. of platinum and in twelve by 0.5 mm. of platinum. The animals have been killed with ether at periods from 3 hours to 170 days after insertion of the radon, and after marking the position of the seed with indian ink the brain has been fixed in formol



FIG. 478.—To show relative sizes of a rabbit's brain, two radon-seed containers 0.3 mm. and 0.5 mm. in thickness, and a radium needle with 0.6-mm. wall.

bromide. Sections have been stained by the following methods: (1) Silver stain for microglia (Del Rio-Hortega); (2) Scharlach R for fat (Herxheimer); (3) Gold for astrocytes (Ramon y Cajal); (4) Bielchowsky (da Fano's modification) for neurofibrils; (5) Weigert-Pal for myelin; (6) Toluidine blue (Nissl) for nerve-cells; (7) Haematoxylin and eosin; (8) Haematoxylin and van Gieson.

Ten Experiments, using Radon Seeds 5 mc., filtered by 0.5 mm. Platinum, applied for 3 to 24 Hours.—The brains which were irradiated for less than twenty-four hours showed no macroscopic changes. Under the microscope, however, vascular congestion was noted after three hours; after six hours fatty degeneration appeared in the endothelium of the blood-vessels, and the irradiated tissue stained poorly, showing as a pale wedge-shaped area with its base on the surface. These changes were more marked by the ninth hour, and swollen microglia cells were visible. At the end of sixteen hours a wider and deeper area of the cortex was involved, and around several of the blood-vessels an extravasation of polymorphonuclear leucocytes was present. After exposure for twenty hours the cortical vessels were congested and there was a cellular infiltration of the meninges. Leucocytes were present in large numbers in the superficial part of the cortex, and there was a well-defined area in which the brain substance appeared necrotic, and was infiltrated with

microglia cells loaded with fat. Surrounding this patch of necrosis was a zone in which the nerve-cells failed to stain for neurofibrils, and the myelin sheaths were becoming swollen and disintegrated.

Previous workers had considered that any changes in the brain tissue proper following irradiation were secondary to hæmorrhage or thrombosis of the cerebral vessels. It must therefore be noted that in the present experiments, although the earliest changes were seen in the vascular endothelium, definite evidence of an effect on the nervous elements, presumably destructive, preceded hæmorrhage or thrombosis.

After irradiation for twenty-four hours a small purplish area was visible close to the seed, and microscopic examination revealed multiple hæmorrhages extending to a considerable depth beneath the surface. The area of pallor was still visible.

The degeneration of the vascular endothelium produced by radium is of very great importance in the treatment of tumours. Some tumours are not only very vascular, but their vessels are thin-walled and poorly supported by the soft tumour substance, and the accident of hæmorrhage into the tumour following irradiation is readily explained.

Twelve Experiments, using Radon Seeds 5 mc., filtered by 0.3 mm. Platinum, applied for 3 to 170 Days.—Two of these were control experiments. In one rabbit a platinum container without radon was placed upon the dura and left for 118 hours. No macroscopic lesion was to be seen, and microscopic examination revealed no changes in the blood-vessels or brain-cells. In the second control experiment the rabbit died under the anæsthetic just after the seed (5 mc.) had been placed. The seed was left in position for twenty-four hours and then the brain was removed. On examination no lesion was found—a result which is only to be expected considering that the lesions are manifestations of vital tissue reaction.

The other ten animals were killed after the radon had been in position for 68 hours, 94 hours, 118 hours, 143 hours, 212 hours, 40 days, 47 days, 54 days, 61 days, and 170 days. After 68 hours an obvious lesion had been produced. There was no intradural blood-clot, but the cortex was plum-coloured for a distance of 2 mm. around a central white streak corresponding to the position of the seed (*Fig. 479*). Section of



FIG. 479.—To show hæmorrhage produced by 5-mc. radon seed, filtered by 0.3 mm. platinum, applied for 5 days.

the brain showed that the discoloration extended for 3 mm. into the brain substance. As the time of exposure was increased up to 212 hours the zone of discoloration increased in size till its diameter measured 8 mm., and the dura became more and more adherent to the damaged cortex. Under the microscope the white area proved to be a mass of necrotic tissue infiltrated with microglial cells loaded with fat. The plum-coloured zone showed multiple small hæmorrhages which after irradiation for 68 hours

extended to the white matter, but after more prolonged irradiation reached the lateral ventricle, and after 212 hours were to be seen even on the far side of the ventricle (*Fig. 480*). All specimens showed fatty changes in the endothelium of the blood-vessels, and after 94 hours thrombosed vessels were found. Microglial cells containing fat were present in the perivascular spaces around the lesion, and were to be found in the subarachnoid space at a considerable distance from it. In the zone surrounding the necrotic area the nerve cells stained poorly, the neurofibrils being fragmented; the astrocytes were becoming disintegrated; and farther away from the lesion the myelin sheaths stained more faintly and showed some segmentation. The actual size of the lesion increased gradually until the 212-hour period, by which time the radon had become almost inactive.

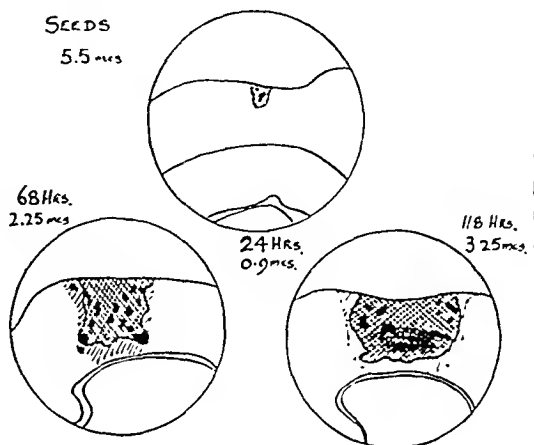


FIG. 480.—Drawings to scale to show the increase in extent of hæmorrhage and necrosis with increase in period of action of radon seed.

When a long period—40 to 170 days—was allowed to elapse before the brain was removed, the purplish area was seen to be replaced by a yellowish-white

core of necrotic material surrounded by a narrow ring of a deep pink colour (*Fig. 481*). Microscopic examination proved the central mass to be necrotic, infiltrated with microglia cells, and walled-in by a ring of proliferating astrocytes which are evidently taking part in the process of repair. As time went on the central necrotic mass became smaller and smaller and the astrocytes more numerous (*Fig. 482*). It must be noted that irradiation can give rise to the formation of astrocytes, and it would therefore be unlikely that irradiation would have a destructive effect on the tumours of which the cells resemble most closely the astrocytes—namely, the astrocytomas. This agrees with the therapeutic results obtained up till the present time.



FIG. 481.—White indurated area of necrosis resulting from application of 5-mc. radon seed, filtered by 0.3 mm. platinum, for 47 days.

It must be concluded from this series of experiments that a large dose of radon can produce a local necrosis of cerebral tissue associated with hæmorrhage; that this process is limited to

parts within a radius of 5 mm. from the seed; and that destruction continues for about 212 hours, being followed by a process of repair. That increasing

the filtration diminished the amount of destruction of brain tissue is shown by comparing the lesions produced by the same dose of emanation (3.2 mc. destroyed) from two 5-mc. seeds, one platinum container being 0.3 mm. thick and the other 0.5 mm. thick (*Fig. 483*).

It must be understood that all the results described so far were produced by very large doses of radon; the effect of therapeutic doses will be described later. The strength of the most powerful seed inserted was 16 mc., and after 116 hours this had produced the only subdural hæmatoma in the series, and the area of discoloured and friable cortex was 1 cm. in diameter. The animal, however,

FIG. 482.—Drawings to scale to show areas of necrosis resulting from application of 5-mc. radon seeds for periods of 40, 47, 54, and 61 days.

showed no sign of involuntary movement or paralysis, and did not appear to suffer at all from this enormous dose. The situation of the lesion probably accounts for this absence of symptoms; for it has been shown by Cairns and Fulton,⁹ again using radon in amounts greatly exceeding the therapeutic dose, that, when applied to the spinal cord of cats, large doses of emanation may produce paraplegia. A large area of the cortex may be destroyed without producing the harmful effects of a small lesion in the conducting paths of the spinal cord.

Seven Experiments, using Radon Seeds 1.5 mc., filtered by 0.5 mm. Platinum, applied for 5 to 21 Days.

—At the end of seven days there was no macroscopic lesion, and histologically there was no clear evidence that any damage had been done. After fourteen days there was a faint blush just beneath the seed, but no macroscopic hæmorrhage. Sections showed tiny superficial hæmorrhages, fatty changes in the walls of the blood-vessels in the immediate vicinity of the seed, and swollen microglia cells containing fat migrating laterally over

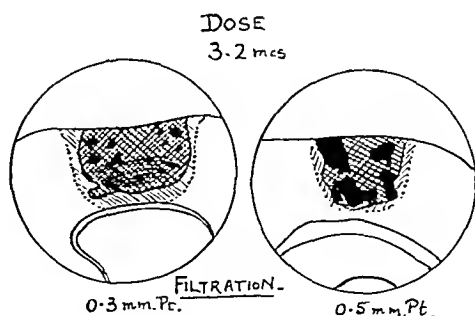


FIG. 483.—Drawings to scale showing effect of the same amount of radon filtered by 0.3 mm. and 0.5 mm. of platinum.

the surface of the brain. The microglia cells and the astrocytes close to the seed were poorly stained, and the processes of the latter were swollen and fragmented, but there was no massive necrosis. No further changes were found after twenty-one days. From these experiments it may be concluded that radon in the strength used therapeutically in other regions produces changes in cerebral tissue which, though of a destructive nature, are not severe enough to contra-indicate the placing of such radon seeds in the questionable, possibly healthy, area around a brain tumour.

Twelve Experiments, using Radium Needles Containing 0.5 mgrm., filtered by 0.6 mm. Platinum, applied for 3 to 58 Days.—The effect of the radium salt itself, distributed so that there is roughly 1 mgrm. of the element for every centimetre active length in platinum needles 0.6 mm. thick, is even less damaging to the rabbit's brain tissue than radon, for in ten out of twelve brains irradiated hæmorrhage did not occur, and in the remaining two it was visible only under the microscope, very minute extravasations having taken place. There were, however, quite definite changes close to the needle, giving clear evidence of a direct action of radium on nerve tissue independent of hæmorrhage.

In a series of nine animals the time of application of the radium needle was increased gradually from three to fifty-eight days. No change was found until the end of a week, when a poorly-stained area was seen around the position of the needle, the nerve elements apparently being more affected than the astrocytes, which stained well. By the end of a fortnight these changes were better marked and extended over a wider area, and fatty changes in the vessel walls, and microglia cells loaded with fat, were visible in the lesion. Even after the longest periods of irradiation the most marked feature was

poor staining, indicative of necrosis of the tissue close to the needle, but there were no hæmorrhages—some of the vessels may have been thrombosed, but the evidence of this was indefinite—and the astrocytes were proliferating around and even within the lesion.

Three animals were treated for twenty-one days and the radium was then removed. The animals were killed four weeks, six weeks, and three months after removal of the radium. These brains showed the destructive lesion of the cortex quite distinctly, and at the site of the lesion the cortex was thinned, and densely infiltrated with astrocytes (*Fig. 484*), forming a scar adherent to the dura, and drawing the deep layers of the cortex and the tracts towards the surface.

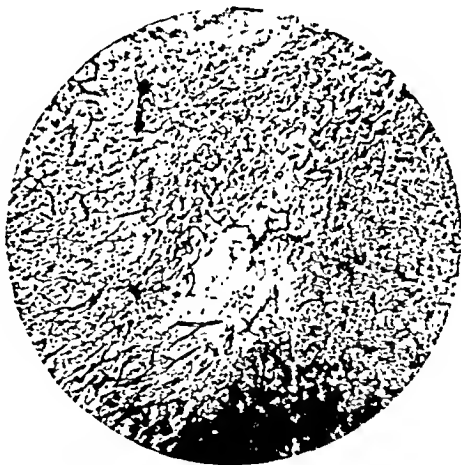


FIG. 484.—Proliferation of astrocytes around lesion produced by extradural application of radium needle for 21 days, animal being killed 3 months after removal of radium.

It may be concluded from these experiments that radon seeds of 1.5 mc. filtered by 0.5 mm. platinum, or needles containing 1 mgrm. of radium element per centimetre active length filtered by 0.6 mm. platinum, acting for seven to fourteen days, have an effect which, though destructive in nature, does not cause serious damage to the cerebral cortex of the rabbit. There is no reason to suppose that such concentrations of radium or radon suitably distributed would be more harmful to human cerebral tissue. It must be noted, however, that one of the results of irradiation is proliferation of astrocytes, a reaction which looks like a response to injury. It may be that some gliomas are a form of reaction to injury. Whether this is so or not, it would appear unreasonable to expect radium to have a beneficial effect on the gliomas the cells of which approach the astrocyte in type.

METHODS OF USING RADIUM.

Having determined as far as possible the dose of radium which may be employed with safety, the next problem concerns the possible methods of applying radium to a cerebral tumour. It must be understood that the blind application of a large dose of radium to the surface of the head in the vicinity of a tumour is unjustifiable. In all cases an attempt must be made to find the tumour and to remove it as far as possible, or at least to determine its nature. A decompressive defect is then to be left in the skull.

If radium is used blindly, a certain number of removable tumours, cysts, and other conditions which yield to surgical attack and will not respond to irradiation may be overlooked. Also radium, in common with X rays, causes an initial congestion and swelling of the brain tissue, which, unless a decompression opening is present, may produce serious or even fatal complications. Operation is therefore the first step, and the further possibilities are:—

1. *Implantation*: (a) Implantation of a large dose of radium in the centre of the tumour; (b) Barrage by multiple small sources of radiation surrounding the tumour.
2. *External application*.

1. *Implantation*.—

a. Implantation of a Large Dose into the Tumour.—At first sight this might appear to be the best plan, for theoretically the radium would destroy the cells of the tumour in its immediate vicinity, and if the tumour were not too large, the outlying parts would stand a good chance of being irradiated also. But a large dose can be left in position for only a short time, owing to the risk of hæmorrhage; and it was formerly the practice to insert 50 mgrm. of radium, heavily filtered, and leave it in position for twenty-four hours. It is now recognized that as a rule the brief application of a concentrated dose of radium is an inferior method of irradiating neoplastic tissue, and it has been abandoned. Occasionally brilliant results followed the use of radium even when so used, but it is doubtful whether the success was attributable to the radium or to surgery. Sir Percy Sargent has kindly permitted me to mention a group of cases treated some years ago by this method. The patients were followed up by Dr. Carmichael, and out of 22 only 3 were doing well—eighteen months, two years, and three years respectively after

operation. The remainder seemed unaffected by the radium, and it is questionable how far the radium was responsible for the successes. For these reasons Sargent altered his technique, and his present practice is described in a recent article in the *BRITISH JOURNAL OF SURGERY*.¹⁰

b. Multiple Foci Surrounding the Tumour.—This is the mode of attack which has proved most satisfactory in the treatment of tumours in other parts of the body. In the case of brain tumours radon seeds or radium needles may be employed. The advantage of radon seeds is that they may be left in position, whereas a second operation has to be performed to remove radium needles. On the other hand, if radium needles are used, it is likely that the irradiation of the area to be treated will be more uniform and complete. If this proves to be the case, then the more accurate distribution of radium in needles may give results which will justify the risk attendant upon a double operation.

Even though a brain tumour be radio-sensitive, the placement of radium so as to deal with it effectively may be a matter of great difficulty. The only satisfactory method of determining the extent of a tumour is to remove as much of it as possible. When this has been done the radium may be placed in the apparently normal brain tissue around the cavity which remains. It may be argued that if this course is followed it cannot be said that the tumour is being treated by radium. It is not to be expected, however, that a large tumour would be destroyed by surrounding it with radium; and it is particularly in dealing with the invisible remnants of growth after 'removal', which produce the 'recurrences' after operation, that the action of radium is required. It must be clearly understood that at present the treatment of cerebral gliomas by radium alone cannot be considered; radium can be used effectively only as an adjuvant to surgery.

This combined method of attack renders the estimation of the efficacy of radium extremely difficult, and it will not be known until patients have been followed up for some years, or until sufficient irradiated tumour tissue has been obtained post mortem. The problems that have to be considered are illustrated by the following cases which have been under the care of the Surgical Professorial Unit at St. Bartholomew's Hospital:—

A man, 36 years of age, had suffered for five years from epileptiform attacks involving mainly the left side of the body, and more recently had noticed that his left leg was growing progressively weaker. The fits were becoming more frequent and were associated with severe headache. Examination revealed evidence of a lesion of the right Rolandic area and bilateral papilloedema. On Nov. 1, 1928, an osteoplastic flap was turned down and the pre-Rolandic convolution was seen to be widened, and at its superior extremity a reddish mass of tumour was seen on the surface. The convolution was explored with a needle, the consistency of the brain was found to alter at a depth of $1\frac{1}{2}$ cm., and on passing the needle further some yellow fluid was obtained. As it seemed unwise to run the risk of damaging this area of the cortex by exploring further, a portion of the tumour was taken for section, and seven radon seeds (2 mc. each, screened by 0.5 mm. platinum) were inserted. The man is now at work, two years after the operation; the left leg is still weak, but the decompression opening at the base of the bone-flap is never tense, there is no papilloedema, and he does not suffer from fits. The tumour, however, was an astrocytoma protoplasmaticum, a glioma the life-history of which is believed to extend over many years, and we have not any evidence at present that the insertion of radon was of importance in relieving this patient's symptoms.

The fallacy of attributing the success of an operation to radium is illustrated by the following case:—

A woman of 52 for a year had been unsteady in gait, and for six months had frequently been unable even to sit up without losing her balance. She was found to have signs pointing to a lesion of the left side of the cerebellum, with internal hydrocephalus and papilloedema. The cerebellum was explored on June 15, 1928, and a vascular papilliferous tumour was discovered projecting between the left cerebellar tonsil and the side of the medulla. A fragment was removed for histological examination, and one seed containing 3.2 mc. of radon filtered by 0.3 mm. platinum was inserted. The tumour was a papilloma of the choroid plexus of the fourth ventricle. The patient made a steady recovery and was able to return to her household duties. In November, 1928, she was shown at a meeting of the Society of British Neurological Surgeons and was regarded as cured. In June, 1929, she returned with abdominal symptoms. The suboccipital region was not bulging, there was no papilloedema or headache, and there was no return of cerebellar signs. She died with multiple abscesses in the liver secondary to diverticulitis of the sigmoid colon. When the cerebellar region was examined post mortem there was only a fibrous nodule where the seed had been placed, but a much larger portion of the papilloma remained in the fourth ventricle. The operation had removed the obstruction in the circulation of cerebrospinal fluid, and although the radium had apparently dealt effectively with the portion of the tumour in which it had been placed, the relief of symptoms cannot be attributed to that.

Another case shows the difficulty of treating a tumour the outline of which cannot be determined, but it also affords important evidence of the efficacy of radium in arresting the growth of a tumour whose cells are of an undifferentiated type.

A man, 42 years of age, had suffered for eighteen months from attacks of vomiting, and nine months before admission to hospital had been compelled to give up his work because of giddiness. During the three months before admission to



FIG. 485.—Large portion of tumour, unsuspected at operation, filling fourth ventricle.

hospital in October, 1929, he had suffered from severe headache, and bradycardia had also been noticed. Examination revealed signs of a tumour in the cerebellum to the right of the middle line, and when this region was explored a dark reddish

tumour was found projecting from the fourth ventricle between the lower extremities of the cerebellar hemispheres. A small portion was removed for examination, and as the tumour was very vascular and was closely related to the medulla no attempt was made to remove it completely, but three radon seeds, 1.5 mc. each, screened by 0.5 mm. platinum, were inserted into what was considered to be the remains of the tumour. The patient did well for a time, but died four months after the operation. At autopsy there was no growth projecting downwards from the fourth ventricle, and at first it appeared that the radon had destroyed the growth. It had destroyed the portion which had been irradiated, but on cutting across the cerebellum an unsuspected mass of tumour was discovered growing from the roof of the fourth ventricle and projecting into it, thus obstructing the circulation of cerebrospinal fluid (*Fig. 485*). The tumour showed the structure of a medulloblastoma (*Fig. 486*): and it was possible post mortem to remove the seeds from the



FIG. 486.—Medulloblastoma. Section of tumour before insertion of radon seed.



FIG. 487.—Medulloblastoma. Showing destruction of the tumour tissue and thrombosis of vessels close to the site of a radon seed.

specimen, mark their position with indian ink, and cut sections of the tissue which had been irradiated. These sections showed that the tumour cells had been destroyed in the zone immediately surrounding a seed, and at a greater distance the tissue failed to stain (*Fig. 487*). The effect was very limited in extent, however, and at a distance of about 1 cm. from the seed the tumour showed the characters of active growth.

The lesson to be learned from this case is that radium can destroy cells of this embryonic type, but that its action is purely local. As far as one could tell, there was little reaction in the surrounding tissues, and, although a few foreign-body giant cells were seen, no very active process or mechanism to remove the necrotic tumour cells was made out.

These are the only cases in which we have had an opportunity of examining irradiated tumour tissue. Another case illustrates the danger of hæmorrhage into the tumour after insertion of radium:—

A woman, 53 years of age, was admitted in coma with signs of a left hemiplegia. Her condition was improved by administration of hypertonic salt solution, and operation a week later revealed a tumour coming to the surface just behind the

central sulcus. A small portion was removed for examination, and ten radon seeds 2.9 mc. each, screened by 0.3 mm. platinum, were inserted. This was in May, 1928, when we were using seeds with more radon and less filtration than we use at present. Fourteen days later, after initial improvement, she suddenly became unconscious again, and the wound was re-opened. A large quantity of blood-clot was evacuated from the tumour and the wound was re-sutured. She returned to her home in the country six weeks later, was able to walk to the doctor's house every day for massage, and mentally she was normal. Six months later, however, symptoms began to return, and she died just over a year after operation.

2. External Application.—Following a decompression operation, radium may be applied externally on a helmet of Columbia paste, plastic wood, or rubber, and this method has been favourably reported on. Some patients so treated have shown improvement greater than that to be expected from decompression alone, and cases have been recorded in which a hernia cerebri has diminished in size or disappeared. It must be remembered, however, that radium has an inhibitory action on the choroid plexuses, and the mere recession of a hernia or the relief of symptoms is far from being evidence that the radium has destroyed a glioma. This method may be used when a tumour has been located but cannot be removed, when implantation presents insuperable difficulties or undue risk, or as a prophylactic measure after removal of a superficial tumour. To treat a deep tumour a large dose of radium is required, and, unless great care is exercised, the intervening tissue may be severely damaged. It is questionable whether this method possesses any advantages over X-ray therapy.

I have only once used external application, in the case of a boy 8 years of age who was found to have a spongioblastoma multiforme deep in the left lobe of the cerebellum involving the left side of the pons. Knowing the results obtained by Cairns and Fulton (referred to above), I did not like to insert radon seeds for fear of damaging the long tracts in the pons, though it must be remembered that they used very large doses of emanation in their experiments, and I think seeds of 1.5 mc. would probably be safe. However, having removed as much of the tumour as possible, I closed the wound, and when it was soundly healed radium was applied on a cap of spongy rubber, a total dose of 19,850 mgrm.-hours being given over a wide area. Before operation the child had been semiconscious. He improved rapidly, and was able to walk steadily in six weeks, and on discharge the only remaining signs were weakness of the left VIth and VIIth nerves. He remained well for five months, and, considering the severity of the condition, we were inclined to attribute the improvement largely to radium. But a sudden relapse, accompanied by signs of further involvement of the pons, suggestive of hæmorrhage into a still active tumour, makes one wonder whether the radium had anything to do with the temporary improvement.

Several articles dealing with radium treatment of the gliomas have appeared during the past ten years, but there are very few accounts of series of cases which would help to assess the value of any one method. In America Frazier and Pancoast were among the earliest workers in this field. Frazier is quoted¹¹ as saying in 1926 that, of 125 posterior-fossa tumours treated by irradiation he was unable in a single case to rule out operative procedures as responsible for the clinical results. Pancoast,¹² comparing radium and

X-ray therapy, says that he has an idea, based upon experience, that radium acts a little more favourably on brain tumours than does the roentgen ray. Hyslop and Lenz,¹¹ however, give it as their opinion that high-voltage X ray is better than the radium 'pack' for lesions more than 5 cm. from the surface; but their clinical records show only a small proportion of cases permanently benefited.

Sargent and Cade¹⁰ have given an account of their experiences with implantation and surface application of radium, but, though some of the astrocytomas treated by them remain well, their paper gives no clear evidence of good results with the spongioblastomas. Cairns¹³ has treated twenty cases of intracranial tumour, mostly spongioblastoma multiforme, with radium during the past three years. In his early cases he inserted radon seeds through the decompressive bone defect after effects of raised intracranial pressure had passed off. He abandoned this method on account of the difficulty of distributing the seeds uniformly through the tumour, and practised direct implantation of seeds under vision. This method he considered dangerous in cases of spongioblastoma multiforme, because of risk of hæmorrhage into the remains of the tumour; and recently he has been collaborating with Cade in the use of surface applications. He believes that it is too early as yet to come to any conclusions, but that there is good evidence that radium has a distinct beneficial effect in some of the malignant gliomas in which surgical methods alone have hitherto given such poor results.

SUGGESTIONS FOR FUTURE PRACTICE.

It must be admitted that on the whole the radium treatment of the gliomas has so far been unconvincing or even disappointing; but sufficient work has not yet been done to warrant the expression of a final opinion. Before that is possible cases will have to be more accurately grouped, both as regards the nature of the tumour and the mode of application of the radium.

Whenever possible a tumour should be removed, and when its extent has thus been determined, radium needles should be inserted around the tumour so as to irradiate the surrounding cerebral tissue evenly and completely, the needles being removed about ten days later. If the tumour is irremovable or its extent cannot be determined, an attempt should be made to excise a fragment for histological examination, and then either radium needles may be inserted where the edges of the tumour are believed to be—as determined by alterations in the consistency of the tissue—or radium may be applied on the surface. In such cases, however, and in the case of deep tumours localized by physical signs or ventriculography but not seen at operation, I am doubtful if much is to be expected from radium, and I am inclined to favour X-ray treatment.

In course of time, records of the partially removable tumours treated with radium by a uniform technique will enable us to say whether radium can deal with gliomas of a certain pathological type or not. At present we may say: (1) That we believe that radium in the dosage suggested above may be inserted harmlessly into the brain; and (2) That we believe that at

all events the majority of the gliomas should respond to radium therapy. If these statements are correct, there must be some reason for the disappointment met with in practice. The most obvious explanation would be that radium has been improperly used; but there is another factor which has to be considered. Though the action of radium on neoplastic tissue is not fully understood, it is believed that when a tumour disappears after radium treatment, not only are the tumour cells destroyed by the radium, but the *débris* which remains is removed by the activity of the neighbouring tissues. It is probable that tissues vary in their ability to deal with such irradiated tumour substance, and such variation might underlie the puzzling behaviour of apparently identical tumours when growing in different parts of the body—for example, an epithelioma of the lip or of the skin of the face disappears rapidly when it is treated by radium, whereas a tumour morphologically indistinguishable growing at the angle of the mouth is extremely resistant; and, again, the response of a primary growth is often very different from that of secondary deposits growing in the lymphoid tissue of a gland.

The reaction which takes place in the brain around a hæmorrhage, or in and around a contused area, is very different from that which occurs in the connective tissues of the body; and whereas in the latter a comparatively small amount of scar tissue is all that remains shortly after an injury, in the brain the process is very much slower, and an area of gliosis, or softening, or a cyst, may be the final result.

In the past our attention has been directed to the cells of the growth almost to the exclusion of the medium in which they are growing. On several occasions it has seemed advisable to excise the indurated portion of a breast which sometimes remains after a carcinoma has been healed by radium. When this tissue is examined, it is usually found to consist of fibrous tissue with a few foreign-body giant cells, and there may be no trace of the carcinoma. The neoplastic cells are presumably destroyed by the radium, and their remains are scavenged away by phagocytes and removed along lymphatic channels. In the brain this scavenging process is probably much less efficient. One of our patients who had been treated with X rays for a deep glioma died as a result of a street accident four months later. When the brain was examined an enormous mass of dead tumour tissue was found; the necrosis may have been the result of X-ray treatment, but nothing was found to show that the dead tissue was being removed. We cannot tell at present how important this aspect of the problem may be, and we are trying to test the hypothesis by experiment. Carmichael has been able to grow a tumour (Pearce-Brown tumour) in the rabbit's brain, and we have recently been applying radium to these tumours. We have not made sufficient observations to justify any conclusions, but we are trying to compare the growth of this tumour in connective tissue and in brain tissue, and its reaction to radium in these situations.

Writing in 1927, Sir James Purves-Stewart expressed the hope that in time surgical operations may be discarded in favour of other more efficient and gentler methods, biochemical or physical, whereby we shall be able to melt away growths, no matter how deeply seated or how insidiously

infiltrating. The time is not yet ; but, though the hope that radium might melt away growths in the brain has not yet been fulfilled, much remains to be done before its true value in the treatment of cerebral tumours is known.

REFERENCES.

- ¹ BAILEY and CUSHING, *Tumours of the Glioma Group*, 1926.
- ² CARMICHAEL, *Jour. Pathol. and Bacteriol.*, 1928, xxxi, 493.
- ³ BAILEY and SOSMAN, *Amer. Jour. Roentgenol.*, 1928, xix, 203.
- ⁴ MARTIN, *Ibid.*, 432.
- ⁵ EWING, *Ibid.*, 1921, viii, 497.
- ⁶ BAGG, *Ibid.*, 536.
- ⁷ PENDERGRASS, HAYMAN, HOUSER, and RAMBO, *Ibid.*, 1922, ix, 553.
- ⁸ HORSLEY and FINZI, *Brit. Med. Jour.*, 1911, ii, 898.
- ⁹ CAIRNS and FULTON, *Lancet*, 1930, ii, 16.
- ¹⁰ SARGENT and CADE, *Brit. Jour. Surg.*, 1931, xviii, 501.
- ¹¹ HYSLOP and LENZ, *Amer. Jour. Med. Sci.*, 1928, clxxvi, 42.
- ¹² PANCOAST, *Amer. Jour. Roentgenol.*, 1928, xix, 1.
- ¹³ CAIRNS, Personal communication.

INTERARTICULAR SYNOVIAL FOLDS.

By J. C. BOILEAU GRANT,

PROFESSOR OF ANATOMY IN THE UNIVERSITY OF MANITOBA.

THIS article, with its accompanying illustrations, calls attention to the constancy with which folds of synovial membrane are to be found in healthy and normal joints, to the extraordinary extent to which these folds project between the articular surfaces of bones, and to the fact that they occur at all ages.

That fringes and pedunculated outgrowths of the synovial membrane lining joint cavities may exist in the pathological state is very well known, but anatomical and surgical text-books do not furnish adequate indications of the presence of synovial folds in health. References to them are so lacking in descriptive detail that it may be doubted if their presence can be generally known or their clinical importance appreciated. They are apt either to be destroyed by the incision which opens the joint or else to be unfolded when the joint capsule is put upon the stretch, and so they elude detection. In

fact, for their effective display, the tissues of the joint must have been so fixed and hardened that the folds retain both their shape and position when the joint is opened. Once, however, their existence, nature, and position are appreciated, no difficulty will be found in devising suitable means to display them.

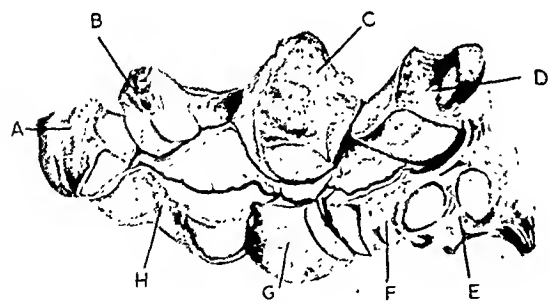


FIG. 488.—Transverse carpal joint, adult, viewed from the palmar aspect. A, Greater multangular; B, Lesser multangular; C, Capitate; D, Hamate; E, Pisiform; F, Triangular; G, Lunate; H, Navicular.

It will be sufficient in the meantime if the existence of these extensive folds be emphasized, for once their existence is known their profound clinical significance becomes apparent. It will not be necessary to do more here than suggest that when a joint is sprained some of its folds are of necessity crushed and bruised, and that the bruising may result in the formation of adhesions, and further that these folds may suffer from various infections as do the valves of the heart. The mechanical and physiological parts they play need not be commented upon just now.

Our attention was first directed to these constant and extensive folds by a dissecting-room specimen of the transverse carpal joint of which *Fig. 488* is a reproduction. When in this particular specimen the joint was opened from the front a broad apron-like fold of synovial membrane was seen to

project from the posterior part of the capsule and to occupy a position between the two rows of bones. The free edge of the fold is thin; its base contains a trace of fat, which is an extension from the fat that occupies the occasional intervals between the fibrous and synovial capsules of this and other articulations. Subsequent dissections showed that, had this joint been opened from the dorsal aspect, a corresponding fold would have been seen to project from the volar aspect of the capsule.

Figs. 489 and 490 show dissections of the left ankle-joint. In each the talus was so removed that any folds of synovial membrane which might intervene between it and its socket (formed by the malleoli and the lower articular end of the tibia) should not be disturbed. In both figures the socket is viewed from below. The fatty pad, marked *X*, is represented in several text-books and atlases, but the folds which are clearly seen to underlie the periphery of the articular surface of the tibia have not, to our knowledge, hitherto been either depicted or clearly described.

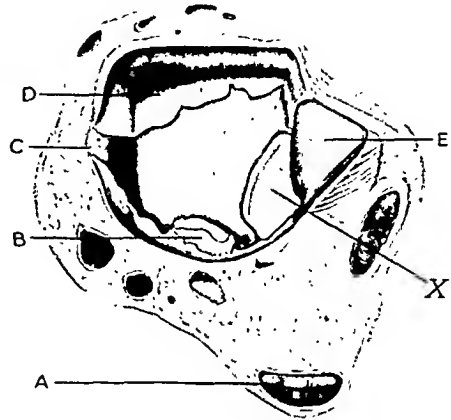


FIG. 489.—Ankle-joint, adult, viewed from below. The talus has been removed from its socket. *A*, Tendo calcaneus; *B*, Folds; *C*, Tibial malleolus; *D*, Fold; *E*, Fibular malleolus; *X*, Fatty pad.

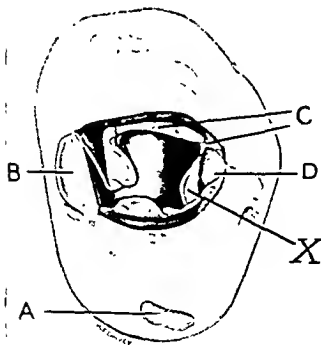


FIG. 490.—Ankle-joint at birth. *A*, Tendo calcaneus; *B*, Tibial malleolus (cut across); *C*, Folds; *D*, Fibular malleolus; *X*, Fatty pad.

Fig. 489 was taken from an adult; *Fig. 490* from a child at birth—and the dispositions of the folds in the two specimens are very much the same.

Figs. 491–493 are of the elbow-joint. In *Fig. 491* the capsule was incised from the front and the collateral ligaments were divided in order to allow the humerus to be gently displaced upwards. This done, the appearance obtained was as depicted—that is to say, folds are seen to encroach on the semilunar notch of the ulna from both sides, and a fold which is almost circular overlies the peripheral portion of the upper aspect of the head of the radius.* This latter fold intervenes between the radius and the humerus much as the semilunar cartilages intervene between the tibia and the femur. In *Fig. 492* this fold is actually composed of fibrocartilage, and might

in this particular instance be referred to as the 'semilunar cartilage' of the

* Several British, French, and German text-books refer to these folds.

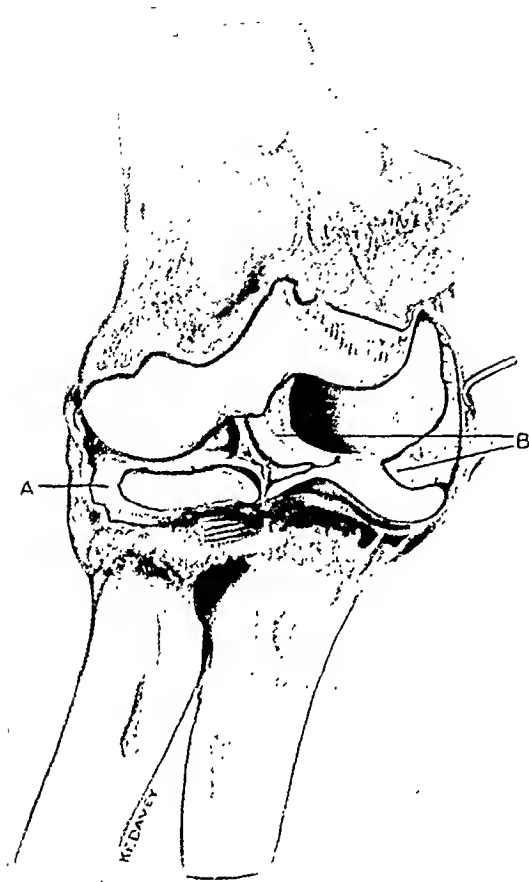


FIG. 491.—Elbow-joint, adult, viewed from in front. A, Circular fold on head of radius; B, Folds.

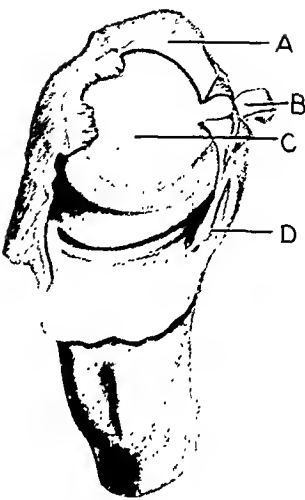


FIG. 492.—Circular fold on head of radius. The fold in this specimen is fibrocartilaginous (adult). A, Fold; B, Portion reflected to show thickness; C, Head of radius; D, Annular ligament.

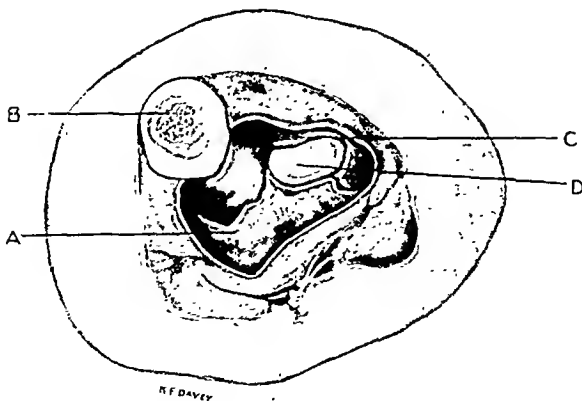


FIG. 493.—Elbow-joint at birth, viewed from above after removal of humerus. A, Fold; B, Olecranon (cut across); C, Fold; D, Head of radius.

radio-humeral joint. The fringes projecting from its anterior end are no doubt pathological.

Fig. 493 is a dissection of the elbow-joint at birth. The capsule was incised all round the joint and the humerus lifted away with as little disturbance to the synovial membrane as possible. The head of the radius and the semilunar notch of the ulna are in this specimen viewed from above. It will be noted that the folds which are present in the adult specimen are present also in the healthy joint of the child at birth, and are, therefore, not the products of age changes occurring as life progresses, neither are they pathological products.

In Fig. 494 the posterior portion of the foot bearing the talus, the navicular bone, and the calcaneus have been removed, leaving the posterior aspects of the cuboid and of the three cuneiform bones exposed. This specimen shows well the characteristic manner in which folds project into healthy arthrodial joints. Under dissecting-room conditions such folds are quite transparent save at their attached margins or bases.

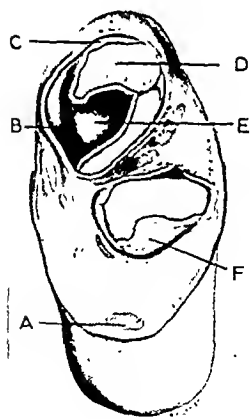


FIG. 495.—Talo-calcaneal and talo-calcaneo-navicular joints at age of 3 years, viewed from above. The talus has been lifted off. A, Tendo calcaneus; B, Spring ligament; C, Fold; D, Navicular; E, Fold on anterior and middle facets of calcaneus; F, Fold on posterior facet of calcaneus.

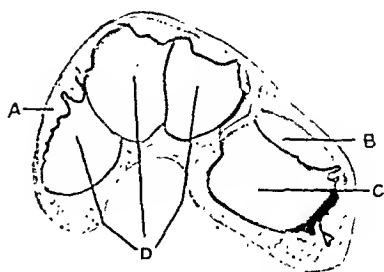


FIG. 494.—Intertarsal joints, adult. The cuneiform and cuboid bones are viewed from behind. A, B, Folds; C, Cuboid; D, Cuneiform bones.

Fig. 495 is taken from a child of 3 years. The talus has been freed and carefully lifted away from the calcaneus and the navicular bone, and, as a result, the extent to which folds project into the talo-calcaneal and talo-calcaneo-navicular joints is well seen. The talus must in part have rested upon these folds. All these folds are present at the time of birth and persist into adult life. The fold, for example, which is seen to overlap the anterior and middle calcaneal facets was present in each of the nine adult specimens we examined.

In brief: in the anatomy rooms our attention was directed to a large fold of synovial membrane which projected far between the two rows of carpal bones and bore their impress. This fold was healthy and not pathological. Investigation revealed its presence in other limbs. Inquiry was then extended in a systematic manner to all the limb joints of the nine cadavera which were then being dissected, the hip-joint alone being unavailable for study. In some dissection had been too thorough for our purpose;

in others the specimens were too dry to be of use. Nevertheless, the material was sufficient to make it perfectly clear that it is the rule for diarthrodial joints (including even such small joints as the intercarpal, intertarsal,

interphalangeal, and superior tibio-fibular) to contain interarticular folds. The illustrations are selected from the various dissections. The ages of the cadavera were 27, 38, 47, 60, 60, 62, 70, 73 and 77 years. The joints of a number of stillborn children were examined, and the joints of a child of 3 years. As folds were found at all ages, and as they are practically the same at all ages, they evidently are not acquired structures, neither are they pathological, but they appear to be necessary parts of the mechanism of joints. That they are of great clinical importance is only too apparent.

The surgeon has for long removed the semilunar cartilage of the knee, and more recently he has removed the posterior part of the humero-radial fold, but that is not to say that the generalization here exemplified is properly appreciated—namely, that all movable joints have at all ages extensive interarticular synovial folds.

My thanks are due to Professor Donald Mainland for checking observations and for collaborating generally in this work.*

* The specimens and the original drawings from which these photographs were made are in the Museum of the Department of Anatomy, the University of Manitoba.

INTRA-VENOUS LOCAL ANÆSTHESIA.

By JOHN T. MORRISON,

SURGEON TO THE ROYAL SOUTHERN HOSPITAL, LIVERPOOL.

Historical Note.—In 1910 I had the privilege of listening to a lecture delivered by Auguste Bier, then professor of surgery at Bonn, in which he expounded the method of intravenous anæsthesia which he had introduced.¹ His technique involved the isolation of an ischæmic section of a limb by means of elastic bandages, the exposure of a vein under infiltration anæsthesia, and the washing out as completely as possible of the whole venous system of that section. Under pressure, 50 to 100 c.c. of $\frac{1}{2}$ per cent novocain solution was then forcibly injected. Anæsthesia of the segment isolated was stated to be immediate and to be 'very satisfactory'. At the end of the operation the venous system was again washed out with saline, the elastic bandages were released, and the wound was closed. Although safe and certain, it can be seen that this technique is tedious and complicated.

In 1909 Goyanes² and in 1913 Braun³ published descriptions of what they called 'intra-arterial' local anæsthesia. After cutting down on 'an artery' under novocain infiltration, 50 to 100 c.c. of $\frac{1}{2}$ per cent novocain solution was injected into the artery, circulation having been arrested by tourniquet immediately before the injection. Anæsthesia came on in 'one to two minutes'. This is somewhat simpler than Bier's method, but still involves an operative interference, which may be far from simple, for the induction of the anæsthesia.

For the last five or six years I have used a method which calls for the use of one tourniquet and one needle puncture only. The underlying principles are somewhat different from those of Braun's and Bier's methods.

Technique.—No time is spent on isolating a segment of a limb. The whole limb distal to the tourniquet is dealt with. There is no advantage in trying to limit the area of anæsthesia, and as a matter of fact it is not possible to do so even by Bier's technique. By means of an elastic bandage or thoroughly padded tourniquet of calibre suited to the bulk of the limb, the arterial circulation is arrested at a convenient level in arm or thigh. Pressure should be applied slowly so as to ensure distension of the veins.* With a fine needle on a Record syringe a superficial vein is entered and a quantity of novocain solution injected. The injection is usually made in the distal direction. A pad and bandage is applied to prevent possible leakage into the tissues. The amount used has varied from 6 c.c. of 2 per cent

* The distended veins are easily seen and clipped before division, and hæmorrhage is no more troublesome than in any other operation.

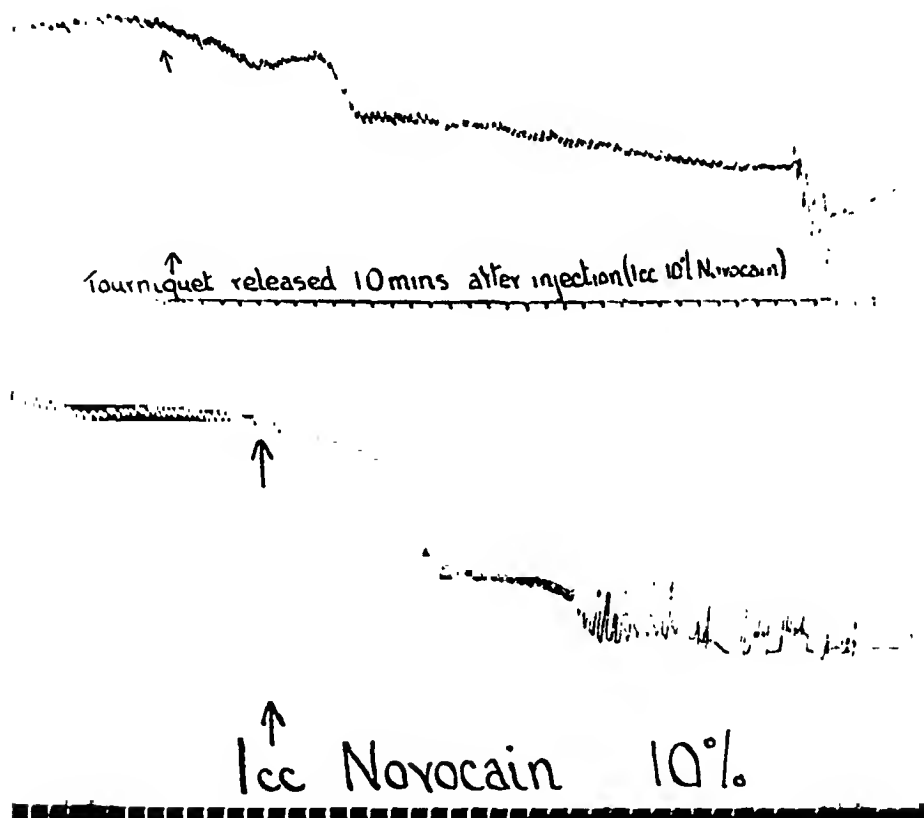
to 12 c.c. of 3 per cent solution, according to the size of the limb to be anæsthetized. The method has so far only been employed in adults. The 6 c.c. of 2 per cent was definitely too small a dose, but 10 or 12 c.c. of 2 per cent is perfectly successful in dealing with an upper limb. The case in which the maximum dose was given—12 c.c. of 3 per cent—was a well-developed though elderly man with a compound fracture-dislocation of the ankle. The injection was made into the dorsal venous arch on the foot and the tourniquet applied above the knee.

In about eight minutes I find that sensation is sufficiently abolished in the deeper parts of the limb to permit of operative interference. Anæsthesia of the skin, however, may not be complete. If this is so, I rapidly infiltrate subcutaneously the line of the incision with $\frac{1}{2}$ per cent novocain and proceed at once. Bier found that, in his method at least, cutaneous anæsthesia of the distal part of the limb was sometimes delayed for as long as twenty minutes. When fully established anæsthesia is absolutely perfect so that even wrenching and dislocating movements give no pain. When the operation is over—but never in less than half an hour after the injection of the anæsthetic—the tourniquet is slowly released so that blood from the limb reaches the general circulation only gradually. This was done in the first instance in fear of toxic symptoms. Bier has shown that novocain cannot be washed out of the limb once anæsthesia has been established, so that apparently care in this direction is not absolutely essential. Nevertheless it is a step in technique which I shall continue to carry out.

Type of Anæsthesia.—The area of cutaneous anæsthesia ultimately extends in many cases as high as the lower edge of the tourniquet, and must therefore be the result of direct action on terminal nerve filaments. In part, however, it is possible that the effects produced are more truly to be described as a regional anæsthesia caused by the drug reaching nerve-trunks comparatively near the site of injection. That this is likely is shown by Bier's cases, in which anæsthesia of the distal portion of the limb occurred, even although his own work proved that no novocain could reach the parts beyond the elastic bandages. The upper limit of deep anæsthesia is not easy to determine. It would seem from Bier's cases that it does not fall far short of the level of the upper tourniquet. In my cases, as it has happened, no suitable occasion for testing this point has arisen.

To what distance novocain actually travels in the dilated (but stagnant) venous channels is perhaps impossible to determine. The relatively high concentration of the solution injected would make it likely that diffusion will play a considerable part. It has been shown experimentally by Petrow⁴ that this does actually occur. After ligaturing a rabbit's ear a small quantity of $\frac{1}{2}$ per cent indigo-carmin was injected into the lateral vein. The ear was watched by transmitted light, and "it was easy to see how, despite the arrested blood-stream, the blue-coloured vessels became gradually lighter, while the surrounding tissues became blue. The diffusion process has in one to two minutes quite definitely commenced, and it reaches its maximum in fifteen to twenty minutes." It will be seen that this corresponds closely to the time during which the 'detoxication' of novocain occurs in the following experiments.

During the operation it is noteworthy that the pain from the tourniquet is greatly diminished in comparison with what results when constriction is applied to an unanæsthetized limb. No doubt still greater comfort could be assured by the use of pneumatic pressure, but of the various types of such tourniquets which I have tested none has been sufficiently constant in pressure to satisfy me that there was no risk of leakage of novocainized blood into the circulation during the course of the operation.



FIGS. 496, 497.—Blood-pressure tracings. The wide excursions towards the right-hand ends are caused by artificial respiration.

Experimental Investigations.—By the courtesy of Professor Dilling, of the Department of Pharmacology in the University of Liverpool—to whom I am indebted for much help—Dr. G. Griffith has carried out a series of experiments for me to determine to what degree and at what rate the ‘fixation’ of novocain in the tissues takes place. With a tourniquet so applied as to arrest the circulation in the hind limb of a 5-lb. cat, 1 c.c. of 10 per cent novocain (1·5 gr.) was injected into a distended vein. After ten minutes the tourniquet was released so that the blood from the limb entered the general

circulation. No change follows the actual injection of what is proved to be an exceedingly poisonous dose. After ten minutes there is, however, apparently sufficient novocain left to give rise to quite a serious fall in blood-pressure (*Fig. 496*), as well as a respiratory embarrassment which for a moment gave anxiety. Five minutes later the same dose was injected into the same vein, this time without the tourniquet (*Fig. 497*). There was immediate cessation of respiration and a fall of blood-pressure practically to nil. The animal, it is true, recovered, but only after prolonged artificial respiration by means of direct insufflation of air through a tracheal cannula. To exclude the possibility of cumulative action of the drug, a series of sublethal doses were given to another animal without ill effect.

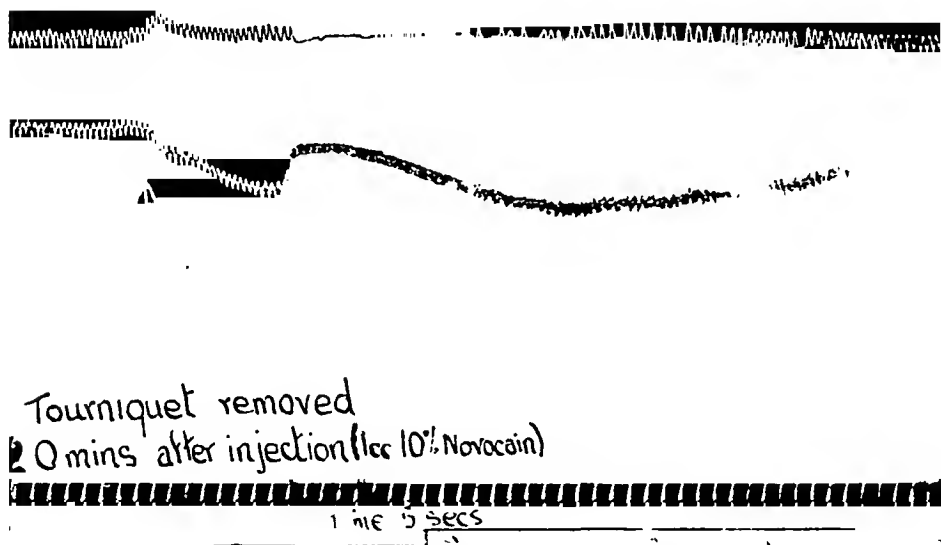


FIG. 498.—The upper tracing is a respiratory record. The fall of blood-pressure immediately following release of the tourniquet is probably due to mechanical causes. It is followed by the more prolonged effect of still 'unfixed' novocain.

The experiment was then repeated as follows. In a slightly larger cat ($5\frac{1}{2}$ lb.) both posterior limbs were isolated by a tourniquet, and an intravenous injection of 1 c.c. of 10 per cent novocain was made into each. After twenty minutes one tourniquet was released, with the very mild vascular and respiratory effects shown in *Fig. 498*. After thirty-five minutes the circulation in the other limb was restored. There was no disturbance of respiration and only the slightest fall of blood-pressure (*Fig. 499*). As it seemed very possible that this minimal response might be due, not to traces of unaltered novocain, but to mechanical changes resulting from the release of the tourniquet, this procedure was carried out without any injection of the drug. The tourniquet was applied as a figure-of-eight round the lower abdomen and upper thigh, and it will be seen (*Fig. 500*) that there is a slight rise of blood-pressure following its application, and a fall when it is released, perfectly comparable to the

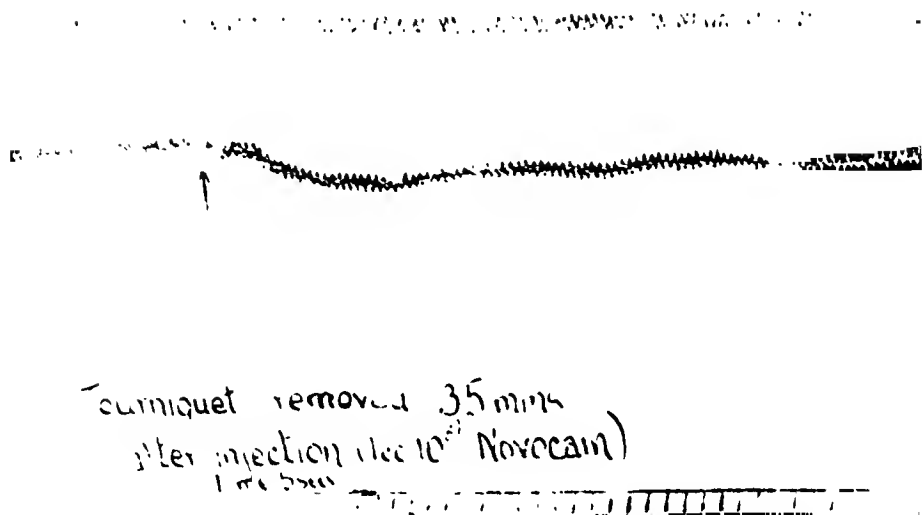


FIG. 499.—Note similarity of changes to those seen in second half of Fig. 500.

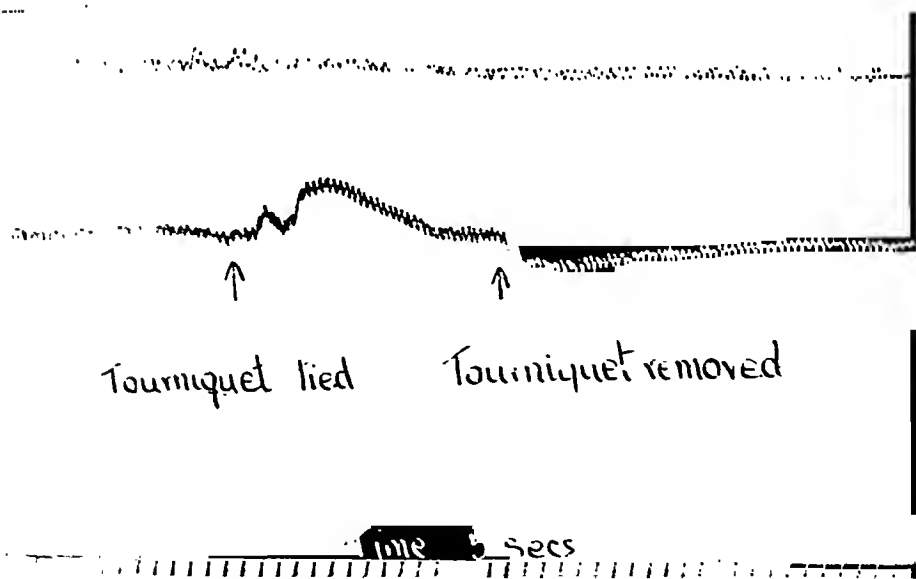


FIG. 500.—No novocain injected; changes in blood-pressure solely due to mechanical effects of tourniquet.

tracing in *Fig. 49J*. It would appear, then, that after the lapse of thirty-five minutes the dose of novocain had in some way become completely innocuous.

A good deal of work has been done in this connection with cocaine, and it is not unreasonable to suppose that the findings may also be applied to novocain. Rifatwaehdini⁵ states that cocaine injected (? intramuscularly) into a ligatured limb and left for several hours is not destroyed, and gives evidence in favour of a process of adsorption having taken place, since the toxicity of the drug is progressively diminished. This latter fact is also noted by Kohlhardt,⁶ who used the intravenous route. Maurel and Pouchet observed that toxicity was diminished by incubating novocain with nervous tissues, but does not say whether this is in any sense a reaction specific to such tissues. There is apparently a very slow release of such 'adsorbed' novocain, but this is not of sufficient importance to make cumulative or delayed action in any way dangerous (Rifatwaehdini⁵). This author's work definitely negatives the conclusions of Wiechowski,⁷ who maintains that cocaine is largely destroyed in the body.

Application of Experimental Results to Man.—The dose employed in these experiments has been 1 c.c. of a 10 per cent solution of novocain. On the basis of the second experiment this is equivalent to a dose of 35.28 gr. for an adult weighing 9 stone. The maximum dose injected in any of my operations was almost exactly 5 gr., so that the margin of safety is very considerable.

Incidentally it has been shown by Dr. Griffith—to whom my best thanks are due—that a dose of $\frac{1}{33}$ gr. per pound weight can be injected into the jugular vein of a cat without untoward effects. Assuming that these results can be applied to man, we find that in a 9-stone individual 3.78 (say $3\frac{3}{4}$) gr. can be safely injected directly into the blood-stream. It is not suggested that such a fact is any justification for carelessness—the more so as individual idiosyncrasies to novocain are well known. Nevertheless, it must come as a welcome relief to the surgeon who fears the possibility of unwittingly making an intravascular injection in the course of an infiltration anaesthesia. It is also to be noted from these experiments that in overdosage, although both fall in blood-pressure and respiratory failure occur, it is the latter which is the more serious and demands the more immediate attention.

Indications.—The following types of case have been dealt with by this method of anaesthesia: (1) Compound fractures and dislocations of the carpal and tarsal bones and joints; (2) Tuberculous disease of the same structures; (3) Tuberculous tenosynovitis; (4) Ganglion of the wrist. In no case have bad results of any kind been seen.* There is no reason why amputations, or indeed any other kind of operative work, should not be done in parts of the body capable of being isolated in this way from the general circulation. In amputations, however, where possible, it would be wise, in order to minimize the loss of blood, to make the limb distal to the operation area ischaemic by means of an Esmarch's bandage before applying the ordinary tourniquet.

* Since writing this paper one case has occurred in which removal of the tourniquet was followed by a momentary syncope and then vomiting. No treatment was necessary, and it is quite possible that the condition was purely psychological in origin.

In the cases in which I have employed intravenous local anæsthesia it has usually been the fear of chest complications in tuberculous subjects that has led to its use. It has also been useful in elderly patients suffering from shock, although in the presence of advanced arterial disease the use of the tourniquet might perhaps be viewed with concern. There can be no doubt that if one were working single-handed it would be invaluable in dealing with lacerations, fractures, or dislocations of the extremities, on account of its simplicity, certainty, and safety. It is not suggested that in the lower limb it should replace spinal anæsthesia, but even there occasions do arise when the necessary solution and a suitable needle are not available for the latter technique.

REFERENCES.

- ¹ BIER, *Edin. Med. Jour.*, 1910, Aug.
- ² GOYANIS, quoted by Bickham, *Operative Surgery*, 1924, i, 214.
- ³ BRAUN, *Brit. Med. Jour.*, 1913, ii, 392.
- ⁴ PETROW, *Zentralb. f. Chir.*, 1909, 482.
- ⁵ RIFATWACHIDINI, *Biochem. Zeits.*, 1913, liv, 83.
- ⁶ KOHLHARDT, *Arch. f. klin. Chir.*, 1902, lxiiv, 927.
- ⁷ WIECHOWSKI, *Arch. f. exper. Pathol. u. Pharmacol.*, 1901, xlv, 155.

NON-PARASITIC CYSTS OF THE LIVER: THEIR CLINICAL AND PATHOLOGICAL ASPECTS.*

By F. D. ACKMAN,

JUNIOR ASSISTANT SURGEON AT MONTREAL GENERAL HOSPITAL;

AND LAWRENCE J. RHEA,

ASSOCIATE PROFESSOR OF PATHOLOGY AT MCGILL UNIVERSITY, AND DIRECTOR OF THE
DEPARTMENT OF PATHOLOGY AT MONTREAL GENERAL HOSPITAL.

THE following case-history is that of a patient who entered the Montreal General Hospital with the signs and symptoms of acute cholecystitis. She was admitted into the service of Professor A. T. Bazin, who at operation found her illness to have resulted from a hæmorrhage into a non-parasitic cyst of the liver.

CASE REPORT.

HISTORY.—Mrs. M., 61 years of age, for thirty-five years had had recurring attacks of distress following the eating of certain foods. These attacks, which were often months apart, came on at various periods after meals, lasted from one to six hours, cleared up spontaneously, and were characterized by a sense of weight in the epigastrium, eructations of gas, nausea, and sometimes vomiting. They were never accompanied by fever, chills, pain, melæna, or clay-coloured stools. Several weeks before admission to the hospital the patient noticed a 'lump' in the right upper quadrant. This did not increase in size and gave no symptoms until two weeks previous to admission, when she was suddenly seized with a knife-like pain across the upper abdomen. The pain was so acute and so severe that it was not relieved by hot applications or a hypodermic of morphine. This pain, which at times radiated to the back and both shoulders, lasted for two hours, and was accompanied by profuse perspiration, nausea, and repeated vomiting. For several days following there was soreness in the right upper quadrant, anorexia, slight nausea, and general malaise. There were no chills, fever, or subsequent jaundice.

ON EXAMINATION.—On admission to hospital physical examination showed a well-developed, somewhat obese female. There was swelling and tenderness in the right upper quadrant of the abdomen, and Murphy's sign was positive. The liver edge was felt two finger-breadths below the right costal margin, but no mass could be palpated. Red blood-cells 4,800,000, white blood-cells 5400, and hæmoglobin 55 per cent. The van den Bergh reaction was negative. X-ray examination of the gall-bladder was negative. A clinical diagnosis of acute cholecystitis was made.

OPERATION.—Professor Bazin, who operated upon the patient under gas-oxygen anæsthesia, found a dark-bluish globular cyst projecting from the anterior margin of the right lobe of the liver (*Fig. 501*). The free surfaces of the cyst were covered with thickened peritoneum, which in places was arranged in strands. On palpation this cyst was found to extend some distance into the liver substance, and involved

* From the Surgical Service of Dr. A. T. Bazin, Professor of Surgery in McGill University, and the Department of Pathology, Montreal General Hospital, Montreal, Canada.

NON-PARASITIC CYSTS OF THE LIVER 649

the posterior inferior surface of the right lobe, where it extended beyond the transverse fissure. The falciform and suspensory ligaments separated the cyst from the left lobe.

On the superior surface of the right lobe of the liver, and 2½ cm. from the lateral margin of the larger cyst, there was a smaller cyst of a similar appearance. This was punctured with a trocar, and 5 c.c. of clear amber fluid were withdrawn.



FIG. 501.—Drawing of the liver cyst as it appeared at operation. A schematic sagittal sectional view is added to demonstrate the depth and size of the cyst.

when this cyst collapsed. On opening this small cyst it was found to be 3 cm. in diameter, and was lined with a glistening membrane which could not be readily stripped off. A trocar was then inserted into the larger cyst and 500 c.c. of chocolate-coloured, cloudy fluid were removed. This cyst extended 16 cm. into the liver substance, and, like the smaller cyst, was lined with a glistening membrane which was intimately attached to the liver tissue. Under observation the cyst cavity decreased in size.

He attaches a special significance and gives a special place to endothelial cysts. He believes that they include not only most of the multiple minute cysts, but many of the larger ones.

Thöle³ merely subdivides the cysts into two large groups:—

Lymph and blood cysts . Cystadenomata.

He would include the bile-duct retention type exemplified in our case among the cystadenomata.

J. F. X. Jones⁴ has given a much more detailed classification, based upon the origin of the cysts:—

Those of teratomatous and embryonic origin	Bile-duct retention cysts
Pseudocysts (degeneration cysts)	Cystadenomata
Lymphatic cysts (single or multiple)	Ciliated epithelial cysts
Cysts of blood-vessel origin	Cystic degeneration of the liver and kidneys.

This classification is the most complete, possibly the most accurate, and includes practically all the cysts that theoretically could originate in the liver.

Etiology.—We have been unable to find in the literature any definite statements as to the percentage of non-parasitic cysts of the liver that are definitely of congenital origin, though they make up the greater number. This is largely due to the wide divergence of opinion among such writers as Virchow, Leppmann,⁵ Sonntag, Thöle, Jones, and others, as to the origin of these cysts of the liver. The teratomatous dermoids and ciliated epithelial cysts are so rare that they may be dismissed with a word, but they are certainly of congenital origin. Lymphatic cysts, whether single or multiple, large or small, and the blood-vessel cysts, also fall within this grouping. Of the bile-duct retention cysts many are undoubtedly congenital. We believe the cysts in our operative and in our post-mortem cases are of the congenital type. Thöle says that cirrhotic changes in the liver, particularly of the biliary type, undoubtedly account for a number of these cysts. Most of the cystadenomata are considered by the majority of authors to be secondary to cirrhosis. Cystic degeneration of the liver and kidneys is also of congenital origin. This leaves only the so-called 'degeneration cysts' to be considered, and these obviously fall into the acquired group. It seems clear, therefore, that the great majority of non-parasitic cysts of the liver are of congenital origin. The following table shows the congenital and acquired types:—

CONGENITAL	ACQUIRED
Teratoma	Degeneration cysts
Lymphatic cysts	Cystadenomata
Blood-vessel cysts	Bile-duct cysts associated with acquired cirrhosis of the liver.
Ciliated epithelial cysts	
Cystic degeneration of the liver and kidneys	
Bile-duct retention cysts.	

Diagnosis.—The very nature of the non-parasitic cysts of the liver makes their pre-operative diagnosis very difficult—so difficult, in fact, that

some authors, as Leppmann, state that an exact diagnosis can be made only at operation.

These congenital cysts are small at first, and usually increase in size so slowly that the liver and even the adjacent organs adapt themselves to their presence without as a rule producing any signs or symptoms unless complications develop. These are, most commonly, sudden undue pressure on the common bile-duct or one of the adjacent organs; secondary infection; or, as in our case, acute hæmorrhage into the cyst cavity. In our case cyst of the liver was not considered in the differential diagnosis. Occasionally an enlarging cyst may pull upon the peritoneum and cause pain of an indefinite character. Less frequently, a cyst arising from the upper surface of the liver may, upon enlargement, cause compression of the lung and even cardiac displacement, with a resulting intermittent dyspnoea, cardiac distress, or both.

Since the favoured location of these cysts is the antero-inferior margin of the liver, pressure from their enlargement most frequently involves the gastro-intestinal tract. As a result biliousness, gastric distension, eructations of gas, anorexia, nausea, and even vomiting may occur. Constipation, or alternating constipation and diarrhoea, may result from pressure on the transverse colon. Very rarely one of the ureters may be obstructed.

Two cases have been described in which pressure upon the portal vein and inferior vena cava has led to ascites and œdema of the legs. Icterus, due to the obstruction of the common bile-duct, has been present in one or two instances. When it has occurred, biliary infection, and not an infected cyst, has been suspected. Not infrequently, as in our case, the patient may present the clinical picture of acute cholecystitis or cholelithiasis. Sometimes these cysts have given rise to a diagnosis of ovarian cyst, as in Stevens's⁶ case.

The larger cysts are often pedunculated and may show a marked degree of mobility. Downward displacement, however, is limited by the liver attachment. This point has been emphasized as of help in making a differential diagnosis. In general, it may be stated that the effect of non-parasitic cysts on the general condition of the patient depends upon the extent of the functional derangement produced in the liver and the organs about it as a result of pressure.

Pneumoperitoneum followed by an X-ray has been used as a diagnostic method, but this procedure has been disappointing in its results. The trocar, formerly used to establish the nature of the cyst contents, should be condemned as a method of diagnosis, on account of the danger of contaminating the peritoneal cavity with infected material, or the possibility of implanting scolices of the echinococcus. Hoffmann⁷ calls attention to the effect of posture as an aid to diagnosis. He has observed that when pain resulted from peritoneal pull, it disappeared on the patient lying down, while, on the other hand, dyspnoea was often induced or exaggerated. In some of the cases, as in ours, where the diagnosis was established at operation and confirmed by microscopical examination, a tumour was never palpated.

Apart from the question of whether a liver cyst is parasitic or not, numerous other cystic and solid tumours must be ruled out before a definite pre-operative diagnosis can be made. The most important of these are cystic and solid tumours of adjacent organs, particularly the pancreas, kidneys, and

ovaries, rarely the spleen; solid tumours of the liver itself; mesenteric and retroperitoneal cysts, or solid tumours at these sites; hydrops or empyema of the gall-bladder; and tuberculous ascites (Alexander). Hoffmann sums up the possibility of arriving at a definite clinical diagnosis of cysts of the liver with the following postulates:—

SUBJECTIVE SYMPTOMS	OBJECTIVE FINDINGS
Increase of pain on standing, and conversely a decrease on lying down	Restricted displacement of the tumour from above downwards even when great lateral mobility exists
Increase of dyspnœa on lying down.	The position of the tumour in relation to the transverse colon.

It will be readily understood, from the wide variation in size and location of non-parasitic cysts, that few of them will fulfil these postulates. Moreover, a differential diagnosis previous to operation is made difficult on account of the similarity in the gross of the various liver cysts. Even if routine tapping of them was justifiable, this would not help much because we know so little about the finer chemistry of their contents. For these reasons one must, in most cases, be content with a diagnosis of "cystic tumour of the liver".

Treatment.—Any non-parasitic cyst of the liver may require treatment at any time, and this is always surgical. Complications such as hæmorrhage, as in our case, or acute infections, demand immediate operation. Wherever it is possible the cyst should be entirely extirpated. Some authors maintain that even the resection of a portion of the liver may be justified. When a cyst is as large as the one here reported and has the anatomical relations of our case, complete resection would scarcely be advisable. When extirpation is impossible, owing to the nature and the location of the cyst, or inadvisable for other reasons, such as infection, the cyst should be laid open, as much of the wall resected as may be expedient, and the cyst then packed and marsupialized, as was done in our case. If anatomical difficulties prevent this, the cyst may be anastomosed with the duodenum as recommended by Everidge. Operative mortality is quoted by Schaack⁸ as 12 per cent.

REFERENCES.

- ¹ MUTO, M., and HANZAWA, S., "Zur Kenntnis der sogenannten solitären wahren Leberzyste", *Mitteil. über allgemeine Pathol. und pathol. Anat.*, 1903, vi, 153.
- ² SONNTAG, E., "Beitrag zur Frage der solitären Leberzysten", *Beitr. z. klin. Chir.*, 1913, lxxxvi, 327.
- ³ THÖLE, F., *Chirurgie der Lebergeschwülste (Neue Dent. Chir.* 7), Stuttgart, 1913, 87.
- ⁴ JONES, J. F. X., "Removal of a Retention Cyst from the Liver", *Ann. of Surg.*, 1923, lxxvii, 68.
- ⁵ LEPPMANN, F., "Über die echten Cysten der Leber", *Deut. Zeits. f. Chir.*, 1900, liv, 446.
- ⁶ STEVENS, T. G., "Large Cystic Tumour of the Liver Simulating an Ovarian Cyst", *Brit. Med. Jour.*, 1925, ii, 952.
- ⁷ HOFFMANN, C., "Über wahre Cysten der Leber mit besonderer Berücksichtigung der klinischen bedeutungsvollen Cystadenoma", *Mitteil. a.d. Grenzgeb. d. Med. u. Chir.*, 1902, x, 477.
- ⁸ SCHAACK, W., "Zur Frage der nichtparasitären Leberzysten", *Arch. f. klin. Chir.*, 1913, cxxv, 183.

*SHORT NOTES OF
RARE OR OBSCURE CASES*

**A CASE OF INTRACRANIAL AEROCELE OR
TRAUMATIC PNEUMOCRANIUM.**

BY ARNOLD ALCOCK.

SURGEON TO THE GLOUCESTERSHIRE ROYAL INFIRMARY.

W. G., MALE, age 34. porter. was admitted to the Gloucestershire Royal Infirmary under my care on Oct. 25. 1930.

HISTORY.—Six weeks before, about Sept. 13. he fractured his skull as the result of a cycle accident. He was unconscious when picked up, and was promptly taken to the Ross Cottage Hospital. There he was detained for two weeks, and then sent home to Gloucester, apparently cured. Four weeks later, on Oct. 25, his own doctor was called in to attend on account of intense headaches. The doctor advised the removal of the patient to the Gloucestershire Royal Infirmary, and the advice was at once followed.

ON EXAMINATION.—The patient was found to be drowsy, and when roused complained of intense occipital headaches. Attention is drawn to the fact that these headaches were intensified on sneezing. The man vomited shortly after admission. He had incontinence of urine. He had complained of photophobia for the past month. There was impairment of the sense of smell, and slight weakness of the muscles of the left side of the face. Otherwise the cranial nerves and general reflexes were normal. There was a scar on the right side of the forehead, and a fracture of the right frontal bone could be felt beneath. Mr. Niceol, the Ophthalmic Surgeon, examined the eyes, and reported as follows: "Right eye—extensive effusion into the vitreous; no view of the fundus. Left eye—optic disc a little pale, but otherwise eye-ground normal. No suggestion of papilloedema."

Oct. 26.—He was rather more drowsy, but otherwise unchanged: temperature normal, pulse 72, and blood-pressure 110–170.

Oct. 27.—A radiological examination of the skull was made by Dr. Goss, Hon. Radiologist to the Institution. His report was as follows: "(1) There is an intracranial collection of air behind, and under, the right frontal bone. (2) There are two points of fracture of the frontal bone near the middle line, the lower involving both plates of the skull and the right frontal sinus. The upper appears to involve the whole thickness of the skull. The intracranial collection of air is probably entering through the right frontal sinus" (Figs. 502, 503).

As no improvement was apparent in the patient's condition, I performed an exploratory operation on the skull, with the assistance of Mr. Bernstein, surgeon in charge of the Nose and Throat Department.

OPERATION.—A large semicircular flap was turned down over the region of the fracture; a small trephine hole was made, and the fractured area of the frontal bone removed. The dura was exposed, but no collection of air (as was to be expected from the X-ray findings) was found. The bone was nibbled away, upon which the dura bulged in the opening to such an extent as to point to greatly increased intracranial tension. A small incision was then made in the dura. No collection of air was tapped, but brain bulged through the dural opening. The opening in the skull was now extended downwards. A curved grooved director was passed in front of, and under, the frontal lobe above the orbital plate of the right frontal bone. At once there was a gush of air, and the bulging brain collapsed. Finally, a large fragment of the frontal bone



FIG. 502.—Intracranial aerocele: frontal view.

which had been removed was replaced, and the scalp sewn up.

SUBSEQUENT HISTORY.—For three or four days the patient remained in a drowsy state, and continued incontinent. He was, however, free from headaches. About ten days after the operation he regained complete bladder control, and his mental condition approximated to the normal. His blood-pressure at this time was 118–160. He made rapid and uninterrupted improvement, and was discharged on Dec. 3, 'quite well'. On Jan. 8 he was shown at a monthly meeting of the Gloucestershire branch of the British Medical Association, and expressed himself as feeling as well as he had ever done in his life.



FIG. 503.—Intracranial aerocele: lateral view.

Comments.—The interesting points to note in this case are the situation and cause of the acrocele, and the progress of the case with complete recovery. The collection of air was extradural, and underneath the frontal lobe. The air was obviously forced through the fracture in the frontal sinus by the act of sneezing. These cases are usually complicated by meningitis, but in this there was an absence of infection throughout. There was no history of rhinorrhœa. Finally, this complication of fractures of the skull is not described in surgical text-books.

Dr. Goss makes the following observations: "In the light of after-events, the result and findings of the operation, and a careful re-examination of the radiograms, it is obvious that there is a layer of brain substance lying between the vault of the skull and the collection of gas—that is, under the frontal lobe. If I could have recognized this before, rather than after, it would have shortened and simplified the operation."

Mr. Bernstein, in his comments, suggests that the operation could have been improved by an exploration through the frontal sinus and by plugging the opening with Horsley's wax, had the situation of the acrocele been correctly diagnosed before the operation.

A review of the literature relating to cranial acrocele can be found in an article by W. E. Dandy.¹ R. D. Mothersole² also reported a case of fracture of the skull followed by presence of air in the cranial cavity.

I am indebted to Dr. Goss and Mr. Bernstein for their collaboration in this case.

REFERENCES.

¹ DANDY, W. E., *Arch. of Surg.*, 1926, xii, 949.

² MOTHERSOLE, R. D., *Brit. Jour. Surg.*, 1928, xv, 514.

FEMORAL HERNIA IN A BOY OF 5 YEARS.

By ERIC I. LLOYD,

SURGEON TO THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET, LONDON.

Femoral hernia is extremely rare in children, and only one example occurred in three years at the Hospital for Sick Children, Great Ormond Street. During the same period 715 inguinal hernias were admitted for operation. Other statistics¹ put the incidence at 0.7 per cent. Girls are much more commonly affected than boys, and Fraser states that he has "never met with an instance of the error in a male child". Everyone who operates upon a large number of hernias in children meets an occasional femoral example in girls, but it seems worth recording its incidence in a boy of 5 years.

The child when seen at the Princess Louise Hospital for Children, Kensington, was obviously suffering from a large right femoral hernia. Operation was performed on Sept. 30, 1930, by the inguinal route, and a sac nearly three inches long was brought up from the crural canal. It was found to be empty, and was transfixed, ligatured, and excised. The inguinal canal was examined, but contained no sac. Recovery was uneventful.

REFERENCE.

¹ FRASER, JOHN, *Surgery of Childhood*, 1926.

A CASE OF FIBROMA OF THE MESENTERY.

By CECIL P. G. WAKELEY,

SURGEON TO KING'S COLLEGE HOSPITAL, LONDON.

TUMOURS of the mesentery are so rare that they may be looked upon as pathological curiosities. They may assume large proportions without giving rise to any marked symptom or causing intestinal obstruction. The case recorded here is of interest on account of the large size of the tumour, which weighed 4 lb. 2 oz., and yet the patient was caused but slight inconvenience.

HISTORY.—Frederick P., a bachelor, age 54, a decorator by trade, was admitted to King's College Hospital, in April, 1930, complaining of a lump in his belly. He first noticed it about three years ago, when it was roughly the size of a cricket ball. During the last year it has gradually increased in size. There was no pain associated with the lump, but the patient has been

obliged to take salts in increasing quantities in order to keep his bowels open. During the last three months previous to admission to hospital he suffered from indigestion. He has always enjoyed good health, and states that he is perfectly fit except for slight indigestion. He has not lost weight. He lived in Australia for seven years about thirty years ago. There was nothing of importance in his past or family history.

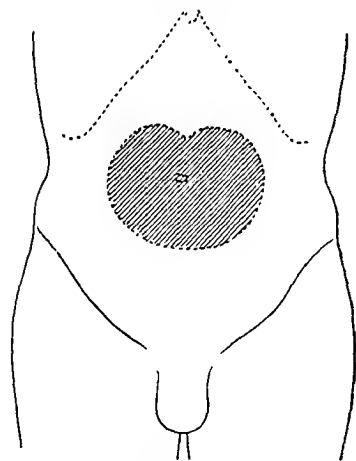


FIG. 504.—Showing the position of the tumour.

ON EXAMINATION.—On examining the abdomen a small para-umbilical hernia was discovered. A large hard tumour could be palpated under the abdominal wall in the umbilical region (*Fig. 504*). It measured about eight inches across and about six inches from above downwards. A small notch was palpable at its upper margin in the mid-line.

The tumour was dull on percussion, but the dullness did not extend into the areas of dullness caused by the liver or spleen. It was hard and smooth, and no thrill or fluctuation could be elicited. It was freely movable in the abdomen in all directions. There was no obstruction to the gut, the patient's bowels were moved each day, and on no occasion was visible peristalsis observed.

OPERATION (April 10).—Operation was performed by my colleague, the late Mr. T. P. Legg. The abdomen was opened by a right paramedian incision eight inches long. On opening the peritoneal cavity a large solid swelling was disclosed lying in the mesentery of the small intestine; one loop of the small bowel was tightly stretched over the middle of the swelling (*Fig. 505*). The loop of intestine involved was about ten feet from the duodenojejunal junction. In order to remove the tumour it was necessary to resect the attached loop of small intestine. A v-shaped enterectomy was performed including the tumour. The two cut ends of the small intestine

were then stitched together, and the cut ends of the mesentery sutured to each other. A drainage tube was inserted down to the line of anastomosis. The abdominal wall was closed in layers.

The patient progressed well for the first twenty-four hours after the operation, when he began to vomit, and his abdomen became distended. A stomach wash-out was given, and 1 c.c. of pituitrin was injected hypodermically. On the fourth day after the operation 20 c.c. of anti-gas-gangrene serum were given. However, the patient gradually sank, and died on the fifth day following the operation.

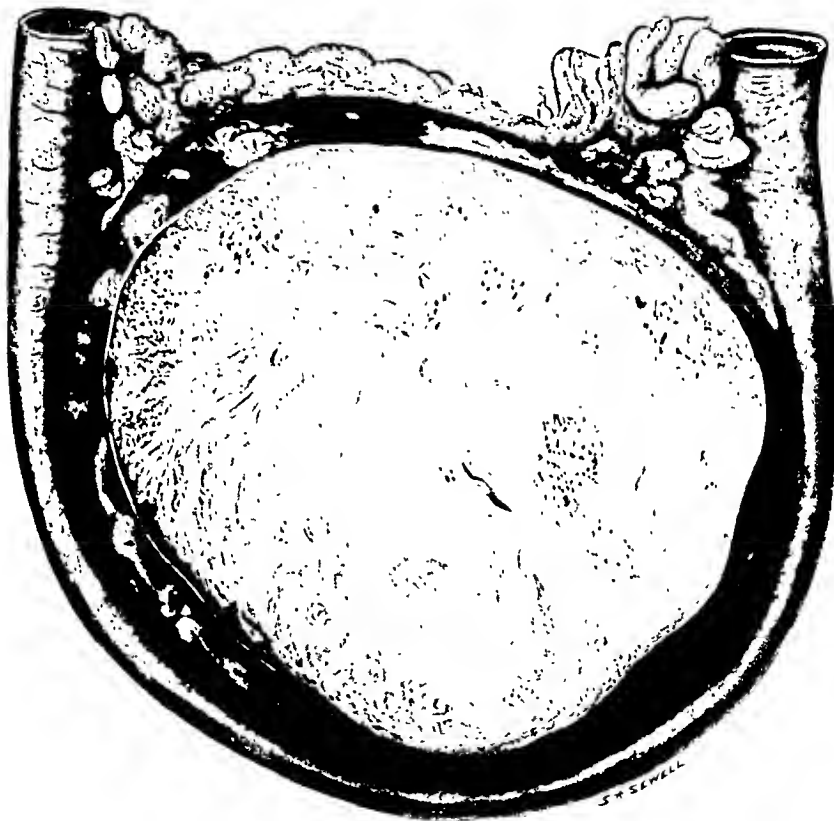


FIG. 505.—The tumour after removal. ($\times \frac{3}{4}$.)

POST-MORTEM.—At the autopsy the cause of death was found to be paralytic ileus caused by a large hæmatoma at the site of the anastomosis. The hæmatoma caused the obstruction and the ileus followed.

The specimen weighed 4 lb. 2 oz., and the cut surface of the tumour was white and hard and characteristic of a fibroma. Microscopically the tumour is a hard fibroma. It is moderately cellular and it is to some extent infiltrated with polymorphonuclear leucocytes and lymphocytes, chiefly near the blood-vessels. The blood-vessels are fairly numerous and are well formed.

A PARTIALLY OSSIFIED FIBROMA OF THE STOMACH WALL.

By H. G. G. NELSON,

SURGEON TO THE ROYAL SOUTH HANTS AND SOUTHAMPTON HOSPITAL.

ON Feb. 1, 1928, I saw in consultation a male of 61 years who had suffered during many years from indefinite abdominal pain.

HISTORY.—The pain was intermittent, had no special relation to the ingestion of food, and there were periods during which he was well, and free from all symptoms. Apart from his gastric disturbance he had always enjoyed good health and had had no serious illness. Fifteen years previously he had what may have been an attack of melæna, but it was not accompanied by hæmatemesis. He had been troubled with eructations and flatulence, but had never vomited. In recent years he had suffered from several attacks of abdominal pain, chiefly in the neighbourhood of the umbilicus; each attack lasted three to four days and was accompanied by slight rise of temperature. When I saw him on Feb. 1, he stated that he had had pain during four days, intermittent in character and localized around the umbilicus. There had been no vomiting, his bowels were regular, though he was in the habit of taking aperients, and the urine was normal. His temperature was 100°, the pulse 80.

ON EXAMINATION.—The patient was a well-built man of a somewhat pallid complexion, but was not anæmic. His teeth were artificial. The tongue was coated. On abdominal examination he referred to the umbilicus as the seat of the pain. The abdomen moved freely on respiration and there was no abdominal distension. There was definite tenderness on palpation over the appendix region, with some relative rigidity. Rectal examination was negative.

FIRST OPERATION.—I considered appendicitis the most probable diagnosis and that the attack was subsiding, and elected to wait before operating. He did not improve, however, and as the pain continued, I operated two days later by a right iliac incision. The appendix was definitely inflamed, thickened, and fixed by adhesions—indubitable evidence of previous inflammatory trouble. Appendicectomy was performed and the wound closed without drainage. I was satisfied that I had correctly diagnosed and treated the trouble.

The patient was doing well, when on Feb. 5, at 8 p.m., seventy-eight hours after the operation, he vomited a small quantity of bright-red blood, and at 10.30 p.m. suddenly collapsed and was obviously suffering from severe internal hæmorrhage. He was treated with intravenous injection of normal saline, the administration of horse serum, etc., and slowly recovered. Two days later there was definite melæna, and a further copious black stool some three days afterwards. I now diagnosed a probable duodenal ulcer, and decided to operate again as soon as his general condition justified interference.

SECOND OPERATION (Feb. 27).—The abdomen was opened to the right of the middle line, in the epigastrium. I was surprised to find no evidence of duodenal ulcer, but immediately felt a lump, apparently movable, situated

in the smaller peritoneal sac above the stomach. Through an opening made in the gastro-hepatic omentum by blunt dissection, the tumour was brought into view. It was white in appearance, somewhat like cartilage but less translucent, round in outline, of stony hardness, and in size larger than a golf ball, though not so large as a tennis ball. It was freely movable, except for an attachment high up on the smaller curvature of the stomach. By careful dissection the tumour was freed and removed. On further examination I found an ulcer on the smaller curvature towards the cardiac end of the stomach. It was impossible to excise this, and I decided that an anterior gastrojejunostomy was the quickest and best treatment; this was consequently performed.

The patient stood the operation well, and through a normal convalescence made an excellent recovery.

PATHOLOGICAL REPORT.—The excised tumour was spherical, densely hard, dull-white in colour, with a smooth surface, but roughened in places where it had been attached to the surrounding structures, and particularly where it had been continuous with the floor of the gastric ulcer. Section of the tumour disclosed in its interior a hard substance, apparently bone. Microscopic examination of the tumour confirmed the naked-eye suspicion of the presence of bone. As seen in the photomicrograph (*Fig. 506*), the main mass of the tumour is fibrous, and clearly delimited within it are masses of bone containing few bone-cells not arranged in Haversian systems, nor has the bone the normal lamellar arrangement. The transition from fibrous tissue to bone in the photomicrograph is abrupt, as if the bone formation had been of long standing in the portions of tumour examined and was not progressing. Within the fibrous tissue masses of cells with an acinar arrangement, whose characters are better shown in *Fig. 507*, indicate an invasion by a glandular structure constituting an adenocarcinoma.

SUBSEQUENT PROGRESS.—It is now (January, 1931) nearly three years since the operation, and the patient remains in good health apart from occasional flatulence. There has been no return of the pain and no recurrence of melaena. He has not lost weight and is able to attend to his daily duties.

COMMENTARY.

The etiology of this curious combination of a mixed epithelial and mesoblastic tumour is not at first sight obvious. Histologically the tumour is a fibroma containing a considerable quantity of fairly well developed regular cancellous bone, and at the same time the tumour is being invaded by a malignant epithelial growth. The fibrous tissue is well formed and regular, and towards the periphery forms a fibrous capsule with definite circumferential limitation. Central to the fibrous capsule, bordering on it, lies the cancellous bone, which has a definition and regularity which is not always present in ordinary heterotopic bone. The epithelial element which invades the tumour is composed of columnar cells, clear and fairly regular in size, though many have two nuclei, and these cells are arranged for the most part in acini or masses. The cells extend between the tumour fibres and into



FIG. 506.—Photomicrograph showing a fibroma of the gastric wall strictly delimited from an area of atypical bone and invaded by an adenocarcinoma. ($\times 110$.)

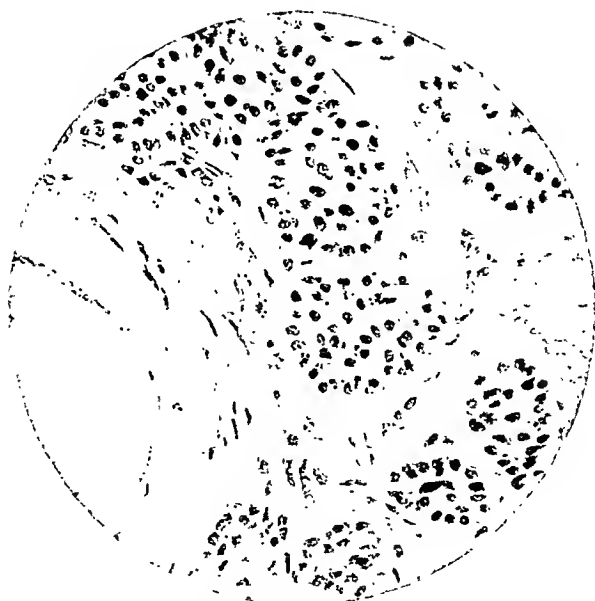


FIG. 507.—Photomicrograph showing the fibrous matrix of the tumour surrounding an area of smooth bone and invaded by cells having the characters and arrangement of an adenocarcinoma. ($\times 300$.)

the cancellous spaces, and this invasion is not now accompanied by any obvious increase of vascularity.

There seem to be three explanations possible—that the tumour may be: (1) A teratoma; (2) A fibroma the seat of an epithelial metastasis; (3) A fibroma being invaded from the stomach by an adenocarcinoma.

1. As regards the teratoma, this might be either primary or metastatic. A primary teratoma attached to the stomach projecting into the smaller peritoneal sac and undergoing an epiblastic malignant degeneration is a possibility, but would be a very rare if not a unique occurrence. Were the teratoma metastatic, its position would be unusual, nor would a metastasis of this size be likely to be single. There is no evidence of a primary teratoma elsewhere—for example, in the testis or in relation to the autonomic nerves or sympathetic ganglia. In the parts of the tumour examined there was no remnant of other somatic tissue besides fibrous connective tissue, bone, and epithelium.

2. A fibroma of the stomach is, of course, a recognized occurrence, and little subserous fibromata, quite apart from anything in the nature of neurofibromatosis, are common; but that a subserous fibroma should attain any great size is certainly not usual. Such a tumour, moreover, would be a pure fibroma, and a carcinomatous metastasis into it without metastases elsewhere would be an extraordinary occurrence. Further, it would not explain the presence of the cancellous bone within the tumour. In any fibroma which had long ceased to grow and had become centrally to a great extent avascular so that the fibrous tissue was functionally dead, calcium salts may be or are ultimately deposited, and if revascularization takes place heterotopic bone may form. But the less vascular the tumour, the less likely is it to become the seat of a metastatic invasion. Therefore this explanation of the tumour's histology seems highly improbable.

3. The third possibility therefore seems the most likely—namely, a fibroma of long duration which has undergone central ossification and has comparatively recently become locally invaded by glandular elements from the gastric mucosa. The gastric symptoms of which this man complained were of at least fifteen years' duration and had at times been in abeyance during many years. It is possible there may have been originally a gastric ulcer with threatened perforation, and therefore with a development about its base of inflammatory connective tissue. It is common knowledge that such inflammatory masses may attain a considerable size, though to form a spherical, almost pedunculated, tumour the size of a golf ball is most unusual. It seems almost possible that the gastric ulcer may have developed—fortuitously perhaps—on the mucosa subjacent to a pre-existing neural or subserous fibroma. That the fibroma, inflammatory or pre-existing, might be invaded by an adenocarcinoma originating in the base of an old gastric ulcer which had recently given rise to attacks of hæmorrhage seems a very feasible explanation. It is not so difficult to envisage an inflammatory fibrous mass being the seat of a calcium deposit as it is to understand this occurring in a slowly-growing gastric true fibroma, though there is no doubt that ossification of true fibromata occurs. The inflammatory fibrous tissue has the property of the cicatrix contracting and diminishing its capillary circulation

and thus rendering itself liable to the deposition of calcium salts. With the epithelial invasion from the stomach, or even before then by simple irritation of the calcareous deposit, a reactionary increase of blood-supply might take place, and, meeting the local excess of calcium, would form heterotopic bone. Though the bone is more regular than heterotopic bone usually is, it seems this explanation best fits the clinical facts as well as the histological findings.

For assistance in the investigation of the tumour I am indebted to Mr. D. M. Greig, Conservator of the Museum of the Royal College of Surgeons of Edinburgh; and my acknowledgements are due to Mr. Tom Hamilton, of the Laboratory of the Royal College of Physicians of Edinburgh, for the photomicrographs which illustrate this article.

TRAUMATIC RUPTURE OF THE AORTA.

By S. J. H. GRIFFITHS,

ASSISTANT SURGEON AT THE GENERAL HOSPITAL, BRISTOL.

RESEARCH into the literature of the last ten years reveals records of a number of cases of rupture of the aorta. The majority are associated with arterial disease—namely, atheroma or aneurysmal dilatation. There are but few

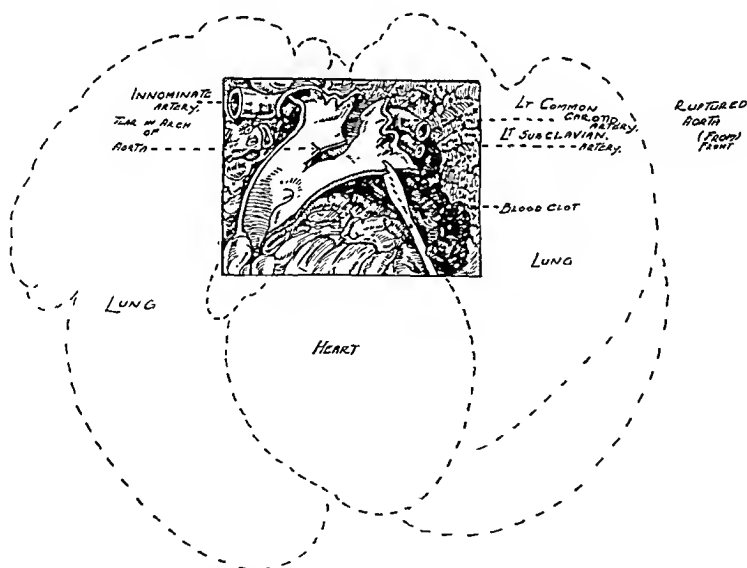


FIG. 508.—Appearance of the aorta post mortem, anterior view.

cases of traumatic rupture, and those described have all been associated with grave thoracic injuries. Kemp¹ described a case occurring in a male of 46 who had been struck on the head and chest by a large stone fly-wheel. The post-mortem examination showed a large abrasion over the sternum, much subcutaneous extravasation of blood around the pectorals, a fracture of the sternum, and a transverse rupture of the aorta just above the aortic valve.

The following case occurring in a young man without any evidence of arterial disease, and with no apparent injury to the chest wall, is worthy of record.

H. A., age 26, was brought in dead to the Bristol General Hospital on Aug. 26, 1929, with the history that he had been involved in a motor-cycle accident.

POST-MORTEM EXAMINATION.—The findings at autopsy were as follows:—

External Appearance.—A young male subject with numerous abrasions upon the forehead and face. The jaw was broken, the nose split, the left tibia and fibula were fractured in three places, and the right wrist was dislocated.

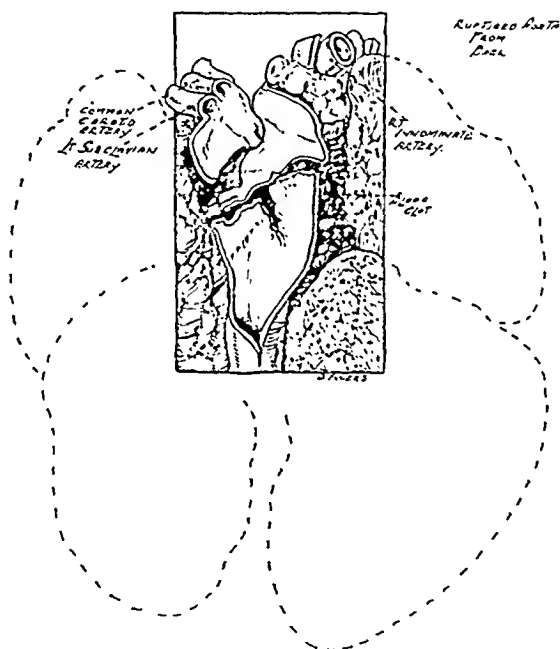


FIG. 509.—Appearance of the aorta post mortem, posterior view.

Internal Examination.—Heart: Myocardium and valves were normal. Lungs: Healthy. Aorta: There was no evidence of disease. At a point beyond the origin of the great vessels there was a T-shaped rupture of the aorta with considerable extravasation of blood around. There was no free fluid in the pleural cavity, no fracture of the skull, and no injury to the abdominal viscera.

There is, I think, but little doubt that this was a case of rupture of the aorta occurring by contrecoup. The diagrams (*Figs. 508, 509*), for which I am indebted to Miss D. Pillers, show the condition of the aorta post mortem.

REFERENCE.

- ¹ KEMP, P. R., *Lancet*, 1923, i, 933.

A THORACIC DERMOID.

By D. E. ROBERTSON,

HOSPITAL FOR SICK CHILDREN, TORONTO.

HISTORY.—The patient, a boy of 7, had always been well until May, 1928. At this date he became ill and a diagnosis of influenza was made. His recovery from this was fairly prompt, but he began to complain of pain in the right chest. The pain came on at intervals, and at times was so severe as to require sedatives. Pleurisy was given as the explanation. This pain, however, ceased after a few weeks and never returned. Toward the end of June, 1928, the mother noticed a swelling of the right side of the chest. The physician considered the bulging was due to fluid in the chest. Throughout the summer of 1928 he was miserable with cough and fever and a considerable loss of weight. Early in December an aspiration was done and some fluid obtained. This was sent to a laboratory for examination and was reported "no T.B."! It is to be noted that this opportunity for a correct diagnosis was missed. Two weeks later, on Dec. 17, a rib resection was done and a cavity drained. The result was not satisfactory in clearing up

the condition, and another operation was undertaken, with no better results. X-ray pictures taken during the foregoing period showed a uniform increased density of the whole right thorax below the second rib (*Fig. 510*).



FIG. 510.—Radiograph of chest before operation.

ON ADMISSION.—He was brought to the Hospital for Sick Children, Toronto, in May, 1929, in an emaciated condition. He had a discharging sinus in the right chest at the level of the seventh rib. The temperature ranged as high as 101° in the evenings, and his pulse-rate was 90 to 120.

ON EXAMINATION.—Examination of the chest revealed a bulging of the right chest with a lack of movement. The bulging was lateral about the sixth or seventh rib, and a sinus was present at the most prominent part. The percussion note was flat all over the right chest except at the apex, where there was a normal resonance to be heard. No breath-sounds were to be heard on auscultation over the area of flatness. It was decided to fill the sinus with an opaque substance and to X-ray the chest, in the expectation of outlining the cavity.

X-ray Examination.—A skiagram was taken after 10 c.c. of sodium iod. solution had been injected into the sinus; the report was as follows:—

On the right side the apex is clear, but from the level of the second rib anteriorly down to the diaphragm very heavy shadows are shown. Most of the dense area lies adjacent to the heart, and separated from the lateral wall by a distance of about one inch except at the sinus, which seems to join it

at the exterior on its postero-lateral aspect. The sinus and the small abscess pocket which it drains are filled with sodium iodide solution. Although the injected cavity and the large dense mass in the centre of the chest appear to be in contact, none of the opaque solution appears to have entered the latter. It is probable that the opaque areas—not injected—are large abscess cavities which are not draining through the injected one.

DIAGNOSIS.—A chronic empyema with incomplete drainage, due to a loculated arrangement.

OPERATION (May 27).—Under general anaesthesia an incision was made in the region of the old sinus, in the mid-axillary line about the sixth and seventh ribs. Incision was deepened to the ribs, where a great deal of new growth had taken place, so that the ribs had fused above and below. A rib resection of about two inches of what was taken to be the seventh rib was done. The region of the sinus was explored with the finger and found to lead posteriorly. Much pus exuded, and at the bottom of the cavity, which had smooth walls, there was felt to be a very hard ridge, smooth and rounded. Anterior to this and between it and the chest wall there was a mound of tissue. A needle was put into this and about 10 c.c. of sero-sanguineous fluid removed. There was great difficulty in determining the nature of the mass. On sponging and cleaning the cavity many hairs were seen to be growing from it. The diagnosis was then clear. The condition of the patient being fairly good, incision in the chest wall was extended considerably and further rib resection done, and a large mass about four and a half to five inches in diameter was delineated in the right thoracic cavity. This was almost round in shape, and could be enucleated fairly readily. The adherent strands to the diaphragm were clamped and cut and tied, as well as many adherent strands to the pleura near the region of the hilus of the lung. The whole mass being enucleated and removed, it was with difficulty delivered through the wound. It was seen that what had previously been taken for an old empyema cavity was a sinus of the cavity inside this mass from which pus had exuded. There was a large space left in the thorax from where the mass had been taken. After all the strands had been tied off and those from the region of the hilus oversewn, no bleeding was noted. Two tape sponges were left in the cavity, the skin wound was sewn up in part of its extent with interrupted silkworm-gut sutures, and left freely communicating with the cavity at its centre. During the latter part of the operation the patient was receiving continuous intravenous saline, and following the operation was transfused with whole blood. His condition was only fair on return to the ward.

Following the operation there was considerable shock, but progress was uneventful. Two days after the operation the large sponges were removed from the cavity. It was clear, but a considerable discharge occurred and persisted for about four weeks. A marked auricular fibrillation occurred two days after operation, but concentrated tincture of digitalis corrected this, and recovery was quite uneventful.

A skiagram taken on May 31, 1929, is reported upon as follows:—

Some large sections have been removed from the sixth and seventh ribs of the right side and a large-calibre drainage-tube has been inserted. The extensive mass which was shown adjoining the right hilum has disappeared,

although there are still some more extensive shadows there than normal. Heavy peribronchial thickening extends upwards into the apex, where there is still some lung functioning. The rest of the right lung is apparently completely collapsed, and a large amount of air fills the cavity in which a drainage-tube has been inserted.

PATHOLOGICAL REPORT.—This report states that the gross specimen consists of a large rounded mass the size of a large grape-fruit, measuring about 12 by 11 cm. in diameter. The mass is definitely encapsulated; the external surface is fairly smooth, except for the presence of a few fibrous tags. Towards one surface is an irregular opening about 4 cm. in diameter, which leads into a cavity in the interior of the mass. Section through the mass reveals that this cavity is of a most irregular nature, having in places a very thin wall about 2 or 3 mm. in thickness, while elsewhere the wall is from 7 to 8 mm. in thickness. The cavity is lined for the most part with a pinkish-white velvety wall, growing from which are numerous fine hairs. In the cavity there is considerable sebaceous material. The wall of the cavity shows great variation in structure. The greater portion of it consists of a yellowish fatty material, elsewhere there are thin pearly-white areas, patches of cartilage (*Fig. 511*).

Microscopic Examination.—Sections were cut from a number of areas showing various pictures in the gross. In these different areas a great variety of tissue structures can be seen. These include connective tissue, muscle, blood-vessels, cartilage, nervous tissue, and epithelial structures. The epithelial structures include sebaceous and coil glands, squamous epithelium, and hair-follicles. In places there are isolated groups of small, deeply-staining cells resembling cells of ependyma. There is no evidence of rapid proliferation or of malignancy.

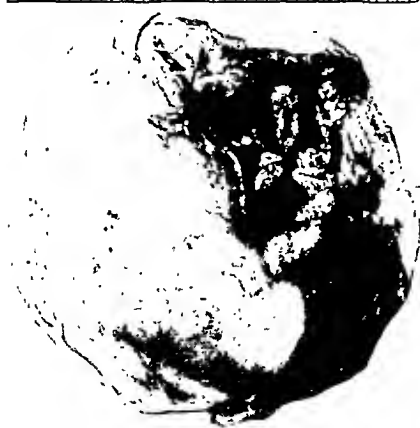
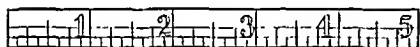


FIG. 511.—Tumour removed from chest, with scale in inches.



FIG. 512.—X-ray appearance of chest six months after operation.

SUBSEQUENT PROGRESS.—The patient's wound finally closed in August, nine weeks after operation. The report of the X-ray taken on Sept. 13, 1929, is as follows:—

Heart and mediastinal structures all appear to be displaced considerably towards the right side of the chest, creating dense shadow in all of the central zone of that side of the chest. The peripheral region of lung appears to be functioning except at the base, where the diaphragm is riding abnormally high and the costophrenic angle is apparently filled up with adhesions.

Dec. 17, 1929.—The lad is now leading a normal life. He has been back at school for six weeks and is apparently well. There is little asymmetry of his chest and there is good resonance over the right side. (*Fig. 512.*)

DISCUSSION.

1. The X-ray pictures did not show the presence of any cartilage or bone.

2. The bronchitis present was mild and no sputum was produced; the breath was sweet, indicating no bronchial opening into any abscess cavity.

3. Deformity and bulging of the chest on the affected side denoted a condition of long standing.

4. A positive diagnosis of dermoid tumour could be made in one of two ways only: (*a*) The fluid obtained at aspiration would have, under close investigation, yielded the correct diagnosis; (*b*) Exploratory opening, with the identification of tissue foreign to the lung.

5. The line of cleavage in this tumour was readily followed. The tumour was so solid that its size could not be diminished by a drainage of any cyst that might have been found in its make-up.

6. A transfusion of blood during the operation was necessary to save this life.

7. The very large cavity left in the right thorax in a boy of 7 required no treatment other than dependent drainage to close in nine weeks.

AN UNUSUALLY LARGE INTRATHECAL SPINAL TUMOUR.*

By LAMBERT ROGERS,

SENIOR ASSISTANT IN THE SURGICAL UNIT, THE WELSH NATIONAL SCHOOL OF MEDICINE, CARDIFF.

This tumour appears to be worthy of record, not only because of its unusually large size, but also because of its position and rapid growth, and the clinical course of the patient before and after its removal. It was removed from a man of 23, weighed 9.5 grm., measured 44 mm. in length, and at its widest part 23 mm. in breadth, and was entirely intrathecal and extramedullary. Originating from the second right cervical nerve, it lay in front and slightly to the right of the upper part of the spinal cord, and extended upwards in front of the cord as far as the foramen magnum. The first symptoms had arisen only four months before operation, at which time the patient was quadriplegic, and its removal was followed by very rapid recovery of function.

* From the Surgical Unit, the Welsh National School of Medicine, the Royal Infirmary, Cardiff.

HISTORY.—In March, 1930, a young marine engineer noticed that he was unable to grip a hammer properly owing to weakness of his right hand. The weakness became greater and gradually extended to the whole arm. Early in May he began to limp because of weakness in the right leg and dragging of the foot. By the beginning of July the right arm was quite useless, the left hand and fingers had become weak, and the right leg stiff. Involuntary contraction, which three weeks before had begun to affect the right leg, now extended to the left leg also, and there was difficulty in commencing micturition. At times he noticed tingling in the right arm, and a feeling of 'pins and needles' in the right leg, especially in the morning just after awakening, but there had never been any pain.

ON EXAMINATION.—The patient was a well-built man with a wasted and spastic right upper limb, adducted at the shoulder and flexed at the elbow. The lower limbs were less spastic and in extension, with Babinski plantar reflexes, ankle-clonus, and exaggerated knee-jerks. The arm-jerks were also exaggerated and the abdominal reflexes absent. He was, with difficulty, able to walk, the gait being not unlike that of a hemiplegic, the right leg stiff, dragging, and circumducted, the right arm hanging by the side and semi-flexed at the elbow.

Pain sensation (pin-prick) was diminished throughout both arms, over the left side of the trunk, the left leg, and the right side of the trunk as low as the twelfth thoracic segment. Over an area corresponding to this distribution also he was unable to distinguish finer differences of temperature. Below the twelfth thoracic segment on the right half of the body and throughout the right leg, however, pain sensation and temperature discrimination were undiminished. There was slight impairment of light touch over the body and all four limbs, and a greater degree in the right arm and hand, the hand being almost anæsthetic. There was not, as might have been expected, any disturbance of the cervical sympathetic. The paralysis, most marked in the right arm and leg, with normal pain and temperature sensations in the same leg, and diminished pain and absent discrimination for the finer degrees of heat and cold in the left leg, constituted a partial Brown-Séquard picture.

DIAGNOSIS.—The Wassermann reaction in the blood and cerebrospinal fluid proved to be negative, and lumbar puncture produced colourless acellular cerebrospinal fluid, the protein content of which was slightly raised. Professor A. M. Kennedy, under whose care the patient was admitted, diagnosed a spinal tumour high in the cervical region.

On Aug. 6 cisternal puncture was performed and lipiodol injected into the subarachnoid space. X-ray examination immediately, and at 6-, 24-, and 48-hour periods afterwards, showed that practically all the lipiodol remained in the cistern. In the 24- and 48-hour plates, only a very small blob, slightly larger in the 48-hour plate than in the 24, was apparent at the lower limit of the meninges. There was thus almost complete blockage of the spinal subarachnoid space at the level of the foramen magnum.

OPERATION (Aug. 14).—Under intrapharyngeal ether anaesthesia, the posterior arches of the atlas and axis and the laminae of the 3rd and 4th cervical vertebrae were removed and the under surface of the occipital bone exposed. On opening the dura mater the appearances were those shown in

Fig. 513. The tumour lay to the right and in front of the cord, which it displaced backwards and slightly to the left. The tumour was crossed posteriorly by a slip of the dentate ligament. This slip was divided, as were also the posterior fascicles of the second cervical nerve on the right side. The tumour was then found to arise in connection with the right second cervical nerve, which was therefore divided at its exit in the intervertebral foramen. The cord was gently rotated, using the cut slip of the dentate ligament, and a large part of the tumour was thereby exposed extending upwards as far as the foramen magnum and for some distance downwards along the anterior face of the cord. After dividing the involved second cervical nerve, the tumour was found to be free, and without difficulty was completely removed in two pieces, which together were found to weigh 9.5 gm. The wound was closed in layers, the edges of the dura mater being only loosely approximated by tying together the sling sutures that had been introduced in order to retract it.

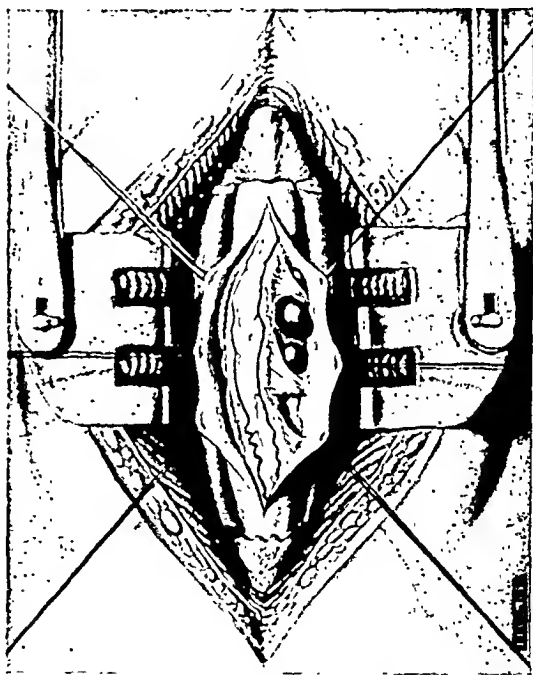


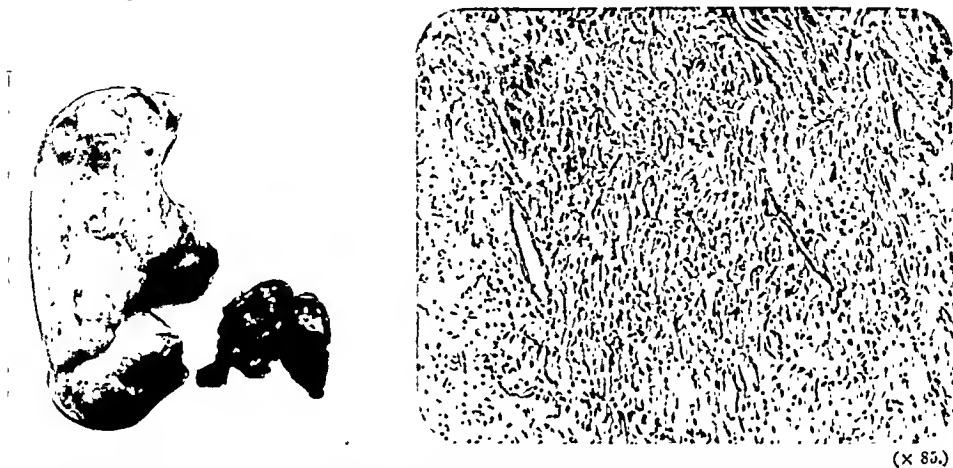
FIG. 513.—Appearance of tumour seen through opened dura mater. The greater part is in front of the cord, and the whole of it anterior to the dentate ligament, a slip of which can be seen crossing behind the tumour between two posterior nerve-roots. The cord is displaced backwards and slightly to the left.

AFTER-HISTORY.—Convalescence was uneventful, primary union occurring. Functional recovery was plain within forty-eight hours of the removal of the tumour, by returning control over the fingers of the right hand. By the fifth day after operation the spasticity had disappeared from the arm, but was rather more marked in the legs. The patient could

now extend the elbow, and finger movements were still freer. Eight days after operation he was able to hold a book in his hand while reading. On the thirteenth day he sat up in bed. Leg movements were now better, and he got up on the eighteenth day. By the twentieth day spasticity had completely disappeared from the legs, and the following day he commenced walking. Pain and thermal sensations had now returned, but there was still some blunting of light touch over the left side of the front and back of the trunk. The man was discharged, walking out unassisted, on the thirtieth day after operation.*

* It is now ten weeks since the operation, and he is returning to his work at sea.

PATHOLOGICAL FINDINGS (*Figs. 514, 515*).—The tumour consisted of loose myxomatous fibrous tissue and was devoid of nerve-cells or nerve-fibres. It was apparently a myxofibroma arising in connection with the sheath of the issuing nerve.



FIGS. 514, 515.—Gross and microscopical appearances of tumour. The smaller part lay to the right and in front of the cord, but in front of the dentate ligament; the larger part entirely in front of the cord. The tumour, which was completely intrathecal, on removal weighed 9.5 grm.

Commentary.—If we exclude those long bedraggled and at times multiple tumours which extend throughout a large part of the spine and are sometimes seen in the lumbar region involving the cauda equina, this is the largest benign, circumscribed, intrathecal, and extramedullary spinal tumour of which I have had personal experience or have been able to find record. The rapid progress of symptoms was not typical of a spinal tumour, and rather suggested tuberculous disease as the cause of the spinal compression. The partial Brown-Séquard syndrome was of interest, because although we should expect this type of dissociation from lateral compression of the cord, it is rather unusual to find it in cases of spinal tumour, the reason probably being that a small lateral displacement of the cord by a tumour results in compression not only on the side of the tumour but also at the diametrically opposite point in the spinal canal, at which the cord is displaced against the bone. By the time the majority of laterally placed tumours manifest their presence hemi-compression has become bilateral compression. The greater diameter of the cervical region of the vertebral canal probably accounts for the occasional presence of the syndrome in cervical spinal tumours. I have seen it in one other case in which also the tumour was a cervical one.

The absence of paralysis of the cervical sympathetic is no doubt explained by the deep position in the cord and the relatively small size of the descending sympathetic fibres.

Following removal of the tumour with release of the compression of the cord the transient increase in spasticity in the legs was typical, and has been seen in a number of cases, but the rapid disappearance of spasticity in the

right arm without any preliminary and apparent increase appears to be unusual. Furthermore, the very rapid recovery of functional activity and sensation is remarkable, and probably the age of the patient, 23, was important in this connection, as in my experience a recovery almost as rapid occurred in a case of a girl of 16, paraplegic and incontinent from a tumour in the dorsal region, who three months after removal of the tumour was able to walk well and had complete control of the sphincters, while in older patients who have made as complete recoveries, up to ten months have been necessary for restoration of function to reach its maximum. The prognosis as regards functional restoration following the relief of spinal compression due to tumour depends upon the degree of damage done to the cord, the position and type of tumour, and the general condition of the patient; but the age of the patient is also highly important, and, other factors being equal, the younger the patient, the better appears to be the outlook to an extent which is much greater than might at first sight be apparent.

I am indebted to Dr. J. B. Duguid, of the Pathology Department of the Welsh National School of Medicine, for kindly reporting on the histology.

REVIEWS AND NOTICES OF BOOKS.

Plarr's *Lives of the Fellows of the Royal College of Surgeons of England*. Revised by Sir D'ARCY POWER, K.B.E. (Mil.), F.R.C.S., Hon. Librarian Royal College of Surgeons; with the assistance of W. G. SPENCER, O.B.E., M.S., F.R.C.S., and Professor G. E. GASK, C.M.G., D.S.O., F.R.C.S. In two volumes. Royal 8vo. Vol. I: Pp. 752 + xxvi. Vol. II: Pp. 596. 1930: Printed and published for the Royal College of Surgeons by John Wright & Sons Ltd., Bristol. Cloth, 42s. net; half bound, 57s. 6d. net.

THESE volumes contain an alphabetical series of biographies of those Fellows of the Royal College of Surgeons of England who have died since the institution of that order in 1843. They form a monumental work with no fewer than 2559 separate notices. The *Lives* is issued as two substantial volumes of 752 and 596 pages respectively, printed on good stout paper with excellent type, and nicely bound in cloth boards with tinted edges and lettering in gilt. The index is most satisfactory, for it gives not only the full name of each entry, but the dates of birth and death whenever that information is available. The plan uniformly followed with each biography or notice has been to record the full name with the date of birth and death and the qualifications or degrees with their dates. What a convenience it would have been if the reader had been relieved of all calculation and the age at death had been plainly stated! Naturally the notices vary much in length, but it appears that all who have left their mark on the history of surgery have been adequately treated. A list of important or principal publications is usually given, and among these there are often additional notes of interest. In each case the sources of information are appended to the notice, and very often interesting and illuminating information is conveyed in this way, while the student of biography is often referred to other works. The *Lives* is much more than a biographical dictionary, for in its 1348 pages is a record of the progress of surgery from the institution of the Fellowship down to but yesterday (1929). In some instances the opportunity has been used to present a summary of the work of a man and its influence on his time, and that is notably so in the case of the life of Lord Lister, where the reader will find an admirable summary, from an easily recognized pen, under the heading of "Antisepsis ensuring Aseptic Surgery", which brings the history of wound treatment down to the commencement of the Great War.

Altogether this is a great work and will remain as a monument to the industry of Victor Plarr and those who revised his notes, brought them up to date, and prepared them for publication. The very title-page is redolent of surgical history and suggests many queries which happily are answered by a perusal of the preface. Who was this Plarr whose self-made monument comes to our table in the form of these delightful volumes? Victor Gustave Plarr, who died in 1929, was librarian to the Royal College of Surgeons for thirty years. The traditions of that office were handed on to him by his predecessor, that great librarian James Blake Bailey, and during his long term the spirit of the place must have become part of his receptive nature, so that when it was suggested to him in 1912 by Sir John Bland-Sutton that he should endeavour to make a record of the lives of the Fellows, the task must have appeared congenial, though sufficiently colossal to have deterred one of lesser spirit. Of course from the outset it was hoped that the lives might ultimately be published, and this idea recurred from time to time, and the collection was examined from that point of view in 1923 when Sir John Bland-Sutton presided over the destinies of the College. Finally, under the presidency of Lord Moynihan, the

collection was prepared for publication by the devoted labours of Sir D'Arcy Power, Mr. W. G. Spencer, and Professor G. E. Gask. But the *Lives* might easily have been buried in obscurity—for is not that the fate of manuscripts "available for reference"?—had it not been for the generous bequest of £5000 made by the late Mr. Thelwall Thomas, of Liverpool, a member of Council, out of which the cost of publication was defrayed. That is the association between Thelwall Thomas and Plarr's *Lives*, and it would be better in a future edition to make it appear so on the title-page.

Many willing workers form the band who turn out a work of this magnitude, and in the preface due acknowledgement is made to the help contributed by those excellent library attendants Fuscaldale and Wood, who, like Plarr, are distinguished by their unfailing courtesy and willingness to help. After the illuminating preface comes a copy of the original Charter. It must be a surprise to those who scan this document for the first time to read in the first sentence, under the heading Charter—in capitals—"that the rank and file of surgeons have always been a wilful and perverse generation". To those of us who suffer under the incubus of that description it is a relief to find that this statement, so startling in its nakedness, is but a prelude to the Charter and not part of that notable document. A rearrangement of the heading on this page would prevent misunderstanding, though it might destroy what is perhaps meant to be the joint-editors' joke! After the Charter follows a list, culled from the *Lancet* of 1843, of the original 300 Fellows, arranged in order of seniority according to date of membership. In this list many notable names appear, and it will be interesting to Fellows not resident in London to notice how many representatives there were from the Provinces. To the list is appended the names of the towns from which the original Fellows were recruited, together with some other particulars of their location. Preceding the actual *Lives* is a statement of the underlying principle which has evidently guided the editors, for we are reminded that

"To calumniate the Dead is Baseness :
To flatter them is surely folly."

*Dr. Johnson, in the Dedication of Charlotte Lemon's
'Shakespeare illustrated'.*

The lives themselves are intensely interesting, and one cannot complain if they appear to be monotonously appreciative, for how can it be otherwise when we learn that for the most part they have been culled from obituary notices published shortly after death? But in very many cases much time has elapsed since then, and recollection or contemporary record might have furnished some *obiter dicta* of the frailties to supplement the catalogue of virtues which are here spread as a feast before posterity. Even a few more personal characteristics would have been welcome and have made these records, excellent as they are, still more human. What a man looks like, the details of his private life, and often the cause and manner of his death, are all alike interesting, though some may think appertaining too much to gossip. But as it is intended to issue a supplementary volume every ten years, corrections and additions are invited, and those who can help to make these records more complete, more accurate, and more intimate are asked to send such particulars as they may have to the Librarian of the Royal College of Surgeons at Lincoln's Inn Fields, London, W.C.2. A few random comments on individual lives may illustrate what is meant.

The notice of Victor Horsley is one of the best in the book, but it is difficult to gather from it even an inkling of his well-known idiosyncrasies. There is no mention of his abhorrence of alcohol, and it would be consoling to some who are less idealistic to know that he was addicted to sweets, which he ate in great quantities. The nicety, even beauty, of his operations on animals will always remain an outstanding memory for those who were privileged witnesses. His kindness to those for whom he took a liking was extraordinary, and many who came in contact with him on active service will remember the many ways in which he endeavoured to help them. It may savour of flippancy to mention that after the funeral of this

very earnest teetotaler and prohibitionist in Amarah on a hot stuffy evening in July, when the temperature during the day had reached 118° F. in the shade, there was a regular stampede to the nearest messes where thirst could be appropriately and pleasantly quenched!

In the notice of Jordan Lloyd, no mention is made of his most valuable paper on "Injury to the Pancreas: A Cause of Effusions into the Lesser Peritoneal Cavity" (*Brit. Med. Jour.*, 1892), which contains practically all that we know to this day about pseudo-cysts of the pancreas. His skill as a surgeon was no more remarkable than the simplicity of his technique, and his Address in Surgery at the British Medical Association in Birmingham was a brilliant and justifiable plea for the many advantages of simple methods. In noting his two residences, it might have been said that he was one of the last to retain consulting-rooms in Broad Street, Birmingham, which he jocularly spoke of as 'the works'.

The notice of Macewen is very good. The barrenness of his classes in the early days probably gave us much of his brilliant work, for he was so devoted to duty that had he been occupied in teaching he would probably not have found the time for those researches which have justly made his name famous. He was accustomed to say that for years he was his only pupil, and in that we of his posterity are probably fortunate. Too little is said of that famous occasion in 1888 when he gave so notable an address before the Glasgow Meeting of the British Medical Association. What he had to say was so novel and at that time so rare, that the audience were as struck dumb until the end, when every man arose to his feet and cheered him to the echo. There is no mention in the article of his *Atlas of Head Sections*, but that was a work which occupied much time and care and deserves to be remembered. As the notice states, Macewen was a tall handsome man; but in the days when he was most sought after his beard was a striking characteristic, though in later years he shaved, which explains the striking difference in his rare portraits. The biography states that his eyes were grey, though the clearest recollection of the reviewer is of a horizon-blue tinge which somehow seemed to add to the peculiar piercing quality and helped to make his face so handsome and arresting when in his prime.

Of Charles Mayo, of Winchester, it has elsewhere been said that "he worked hard every day of his life and continued his hospital duties to the age of 83", which briefly but succinctly emphasizes the achievements of this grand old man. Frederick Page, of Newcastle, was a great personality, a brilliant operator and an entertaining clinical lecturer. He was a small man, quick in every movement, impatient, and often bad-tempered, but none the less kind-hearted and a staunch friend and always appreciative of enthusiasm in others. The note on his life overlooks the fact that in his declining years he found congenial employment in the exercise of his responsibilities as a Justice of the Peace. Sinclair White was a remarkable man. He began life in Sheffield as tutor in the Medical School, then became house surgeon, immediately afterwards Officer of Health, then general practitioner, from which he drifted into surgery, and ended as head of the surgical school of his adopted town! He was a great worker and better known for his success in the art of surgery and for his influence in the school and hospital than for any scientific attributes. In 1906 he acted as one of the Secretaries to the Surgical Section of the British Medical Association in Toronto. This was one of the most active sections of that memorable meeting, and the duties of the Secretary can seldom have ever been more onerous, though White discharged them punctiliously and well. He had a weak, ineffective voice and had great difficulty in making himself heard among any but the smallest audience.

It is interesting to know the manner of a man, and with regard to Henry Brunton Angus it would have been a better word-picture were we told that he was tall and good-looking, in his youth a footballer, and in his more mature years an enthusiastic fisherman. It was also a matter of local historical interest that for some years, in the earliest days of the development of X rays, he was in charge of the then primitive department.

Thomas Annandale was a big, powerful man who in every way cultivated the

style of his old chief, Syme, so that in his later years he came to be in appearance singularly like his old and admired master, with the bushy side-whiskers and the big spotted bow tie. His father, also Thomas, was for some years Honorary Surgeon to the Newcastle Royal Infirmary. The Edinburgh Annandale was wonderfully vigorous, and it was a delight to witness his operative dexterity and his neatness. Arthur Barker was an example, none too frequent in Britain, of a surgeon imported from another school in order to add 'new blood' to a stock which was ready for the stimulus. In the admirable account of this surgeon rather too little emphasis is laid on his articles on spinal anaesthesia, which were most admirable for their great detail and painstaking accuracy, and though the method which he advocated has been superseded, all who are interested in this subject would do well to read what he had to say on the matter. Thomas Bryant was one of the most handsome men of the Victorian era which he adorned, and those who knew him then and have grown accustomed to the rather *négligé* attire of the modern surgeon, must be struck by the difference in the ways of the times. It is sad to think that his declining years were darkened by financial embarrassment, but a relief to know that a civil list pension provided a welcome aid. In the otherwise good account of this surgeon, nothing is said about the early use of iodine in the treatment of wounds, which certainly preceded the modern practice of the use of that antiseptic. The inclusion of the name of Dennis Embleton, so long Nestor of the profession in Newcastle-upon-Tyne, comes as a surprise to those who remember him only as an accomplished and scholarly physician known to most students of the Newcastle school as the author of a history of its inception and early days. Many stories centre round his clinical teaching, and of him, doubtless as of others, it is recorded that in the days before chemical tests were generally used, he taught the students to dip a finger in urine and to taste it in order to detect the presence of sugar! George Yeoman Heath, also of the Newcastle school, was a great figure in his day: a handsome man, a brilliant surgeon, and an absorbing lecturer. Among the students he was noted for unpunctuality, and it is recorded that on one occasion when he arrived an hour late for lecture, all but one had left the precincts of the college. Unabashed, Heath took this student to the lecture room and entertained him for a full hour with a brilliant exposition of the subject in hand. He loved horses and was very proud of his spanking turn-out. Any notice to his memory should state his generous benefactions to the College of Medicine in Newcastle, for he left a sum of £6000 to found a residential hall and endow the chair of Comparative Pathology which is known by his name.

Many of the notices could not be improved, like that on Edward Coek, of whom it is recorded: "As a man Coek was the best beloved of the Surgeons who have been attached to Guy's Hospital. In his youth he was always known as 'Old Coek', in old age as 'Teddy Coek'." There are abundant examples from the same source, and the eponyms of D'Arcy Power and Lord Moynihan's most entertaining article on the earlier surgeons of the Leeds School will always remain as excellent models.

These random observations are merely to show that there are ways in which the admirable notices that form this collection may be made more interesting if not too gossipy. The general excellence of the *Lives* will excuse many little mistakes which have crept in, and the few occasions on which the obviously rough notes of the first compilation have been allowed to stand without that editing which would make them more polished. There are mistakes of commission as well as omission. George Ernest Herman's most useful and successful book was entitled *Difficult Labour* and not *Difficult Labours*. John Macewen is still surgeon to the Glasgow Royal Infirmary. Viotor Horsley died at Amarah on the Tigris, not Amerah. Dexter is the right and not the left side of a picture. But these and many other slips are merely trivialities, and can easily be corrected in another edition. The work is a great joy, and there can be few who will not delight in browsing among the pages, and none who can afford to neglect this mine of information when searching for particulars about the 'Fellows' of yesterday or gleanings of surgical history.

Manual of Surgery for Students and Practitioners (Rose and Carless). By CECIL P. G. WAKELEY, F.R.C.S., F.R.S. Edin., Erasmus Wilson Lecturer, Royal College of Surgeons of England; Surgeon, King's College Hospital, etc.; and JOHN B. HUNTER, M.C., M.Chir. (Cantab.), F.R.C.S., Assistant Surgeon, King's College Hospital, etc. Thirteenth edition. $9\frac{1}{2}'' \times 6\frac{1}{2}''$. Pp. 1592 + xii, with 664 illustrations in the text, 19 coloured, and X-ray supplement. 1930. London: Baillière, Tindall & Cox. 30s. net.

It is only three years since we reviewed the last edition of this well-known textbook of surgery, and we would congratulate its editors on its continued prosperity. Mr. Carless writes what he describes as a valedictory preface, after having been chiefly responsible for the work for thirty-two years. Chapters on special subjects have been written or revised for this edition: bacteriology, by W. E. Carnegie Dickson; ear, nose, and throat, by V. E. Negus; the eye, by N. Bishop Harman; tropical surgery, by Sir Frank Connor; gynaecology, by Eardley Holland; and anaesthesia, by C. F. Hadfield. A new radiographic supplement by Graham Hodgson has been added.

This work has now established so unassailable a position as the first of its kind in this country that any praise for its general excellence and for the diligence of its present editors would be superfluous. If therefore we criticize some details, it is only in the hope of further perfecting future editions.

The statement that in congenital dislocation of the hip no true dislocation exists at birth is not borne out by museum specimens. Further, it is not correct to say that in cases of this deformity in patients over fourteen years of age operative treatment is useless, because very great improvement can be effected by a bifurcation osteotomy. Stabilization of a flail foot is said to be best effected by a subastragaloid arthrodesis, and no mention is made of the necessity of also fixing the mid-tarsal joint as in Naughton Dunn's operation. In fractures about the shoulder-joint we do not think that enough stress is laid upon the importance of abduction, and we think that the modern shoulder abduction splint should take the place of the obsolete Middeldorpf's triangle. In Pott's fracture the need for using a tilted shoe in after-treatment is not mentioned. In fractures of the os calcis the necessity for disimpaction with pin traction on the heel is omitted. In the description of the examination of the ureter and renal pelvis, we think that the use of uroselectan ought to have been included. We are glad that the chapter on amputation has been simplified and modernized and that the use of the guillotine operation is condemned. Among the forty-three excellent radiographs in the supplement, we regret that one should have been included showing a long screw in a fractured olecranon—a method of treatment which is surgically and mechanically bad.

The new edition has a larger page, many new illustrations of great beauty have been added, and the work will long remain most popular among students and practitioners.

The Science and Practice of Surgery. By W. H. C. ROMANIS, M.A., M.B., M.Ch. (Cantab.), F.R.C.S. (Eng.), F.R.S. (Edin.), Senior Surgeon in Charge of Out-patients, Surgeon to the Isolation Block, and Teacher of Practical Surgery, St. Thomas's Hospital, etc., and PHILIP H. MITCHNER, M.D., M.S. (Lond.), F.R.C.S., Surgeon in charge of Out-patients, Teacher of Operative Surgery, and Demonstrator of Anatomy, St. Thomas's Hospital, etc. Third edition. In two volumes, with 716 illustrations. Vol. I, General Surgery. Pp. 772 + x. Vol. II, Regional Surgery. Pp. 950 + x. 1930. London: J. & A. Churchill. 14s. each volume.

WHEN we reviewed the first edition of this work some two years ago we expressed the opinion that it would meet with approval because it appeared to be an endeavour on the part of two actively minded surgeons to record what they had seen and to advocate the treatment of which they had personal experience. It is, therefore, not a matter for surprise—though certainly one for congratulation—that a third edition should be called for within two years. Incidentally, we wonder why the authors depart from the practice, which has always appeared to us as being a good one, of dating their prefaces. We were not privileged to see the second edition, but as compared with the first the third edition is certainly a revision, and some

pages have been almost rewritten. If we do not agree entirely with some statements which are made, it is in general a question of detail, and we have nothing but praise for a certain dogmatism in the description of symptomatology and treatment which suggests that what we read is the personal experience and conviction of the authors. We would suggest that when undertaking the fourth edition the authors should first go through the present volume and cut out everything which they have not seen or done. This would make room for much-needed space for a more complete description of certain diseases, such as acute osteomyelitis, the teratomata, etc. Why do they still perpetuate prehistoric amputations of the lower limbs? Some of these are described under operations of historic interest; and a student, reading for an examination, might realize that this section could be ignored. But on the preceding page, amputations are described which certainly have no place in modern surgery. The figures are not well chosen—in many instances depicting conditions of extreme rarity and of no practical value. The representation of the complete armamentarium for an anæsthetist is surely unnecessary; and some figures, such as those of the ice-tong caliper and the Laue's plate, should be replaced by types in modern use. On the whole we regard this as the best British text-book of moderate size, but apart from this its popularity is enhanced by its extreme cheapness, which may provide an example worthy of imitation by other publishers.

Surgical Emergencies in Practice. By W. H. C. ROMANIS, M.A., M.B., M.Ch. (Cantab.), F.R.C.S. (Eng.), F.R.S. (Edin.), Surgeon to and Lecturer in Surgery at St. Thomas's Hospital, etc.; and PHILIP H. MITCHELL, M.D., M.S. (Lond.), F.R.C.S., Surgeon in charge of Out-patients, Teacher of Operative Surgery, and Demonstrator of Anatomy, St. Thomas's Hospital, etc. Demy 8vo. Pp. 608 + vii, with 158 illustrations. 1931. London: J. & A. Churchill. 18s. net.

THIS book has been produced by the authors of the well-known *Science and Practice of Surgery* to enable the practitioner who, from choice or necessity, has to deal with urgent surgical cases to obtain readily the necessary information in as concise a form as possible. On the whole the authors have succeeded in their object, though in places advice is given which is open to criticism and details are omitted which are of extreme importance. The preliminary chapter dealing with operative technique and pre- and post-operative treatment is clear and informative, and a useful section is included describing the arrangements necessary for an emergency operation in a private house. In the section on injuries to the soft parts special reference may be made to the section on burns and scalds, in which the importance of early cleansing of the burned area, so often neglected, is insisted upon and a definite line of treatment for such cases is provided. It is, however, to be regretted that insufficient stress is laid on the importance of avoiding contractures by their prevention in the early stages. Whitlow, in its various forms, is fully described and the necessity of early incision and of the proper planning of the incision is clearly indicated. The importance of an X-ray in the case of all fractures or suspected fractures from a legal point of view is, or should be, now well known, but the very important point is urged that on no account should treatment of a fracture be delayed because no X-ray is immediately available. Of individual fractures a sufficient account is given of the treatment of each type without unnecessary multiplication of methods. It is, however, disappointing that so little space is given regarding the possibility of the occurrence of Volkmann's contracture and its prevention in cases of injury in the region of the elbow-joint.

The chapters on abdominal emergencies are good, though some exception may be taken to the author's advice on the treatment of acute appendicitis; in the hands of experts, to delay operation, if a case is seen after forty-eight hours from the onset, *may* be good surgery; but to give this advice to general practitioners is bound to cause an avoidable mortality in such cases. The description of the after-treatment of general peritonitis is confusing, and it is doubtful whether it would be of much value to a man who had but little previous experience: a dogmatic statement of what to do in such cases would be of more value than a number of hints.

The blemishes that have been pointed out are outweighed by the excellence of the rest of the book, and it seems certain that it will become a valued addition to the practitioner's library.

Emergency Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London. Volume I. Abdomen and Pelvis. Medium 8vo. Pp. 380 + xx, with 324 illustrations, some of which are in colour. 1930. Bristol: John Wright & Sons Ltd. 25s. net.

THIS is the first volume of a two-volume work upon emergency surgery and is devoted entirely to the abdomen and pelvis. The author's avowed purpose is to assist the "comparatively isolated surgeon" who is suddenly brought face to face with an acute abdominal catastrophe, and, on the whole, we may congratulate him upon having adequately achieved his intention. Precise details are given of each operative procedure described, each is usefully illustrated, and a certain degree of dogmatism, unsuitable in a more systematic treatise, may well be pardoned in a work of this kind. The methods described are fully up to date—in fact, it is a question whether some of the procedures advocated have been in practice sufficiently long to justify their recommendation to a "comparatively isolated surgeon". The 'delayed' (Ochsner-Sherren) treatment is strongly recommended as a routine practice in cases of acute appendicitis of over forty-eight to fifty hours' duration, though it is stated that this "must be carried out on the threshold of the operating theatre".

Leonardo da Vinci, the Anatomist (1452-1519). By J. PLAYFAIR McMURRICH, Professor of Anatomy, University of Toronto. 7" by 10". Pp. 265 + xx, with 89 plates and illustrations. 1930. London: Baillière, Tindall & Cox. 27s. net.

WHEN reading this book one wonders how many of those drawn to the Exhibition of Italian Art of 1930, amid the dazzling delights and masterpieces, sought out the small, comparatively hidden, pen-and-ink sketches of Leonardo. All but one were anatomical, and yet, as one gloated over them, one felt sure that the majority of those obviously interested had no special knowledge of this science.

Professor McMurrich writes as an anatomist of an anatomist, and we have chapters which show the state of anatomical knowledge which served as a basis for Leonardo's work, how he worked, his anatomical manuscripts, and other matters connected with this particular aspect of so many-sided a character. Yet one cannot help thinking that the author was tempted to allot an undue space to the fascinating account of da Vinci's fortunes and his friends, in which we learn of his successive patrons, Sforza, Duke of Milan, Cæsar Borgia, and Charles d'Amboise: here we have the story, with a greater regard to historical accuracy, which forms so charming a basis of *The Forerunner* by Merejkowski.

The question naturally arises whether Leonardo, had he been less interested in other arts, would have been a greater painter. Without doubt his output which, compared with the colossal activities of some of his predecessors, was so small as to suggest indolence, would have been far greater had he given painting his undivided attention; yet it may be that a number of works of less merit would have less ably sustained his reputation than the few of which "The Adoration of the Magi" may be regarded as the type. As an anatomist he will be known to posterity as the discoverer of the moderator band in the heart; as an anatomical illustrator he was the first to depict what he actually saw in a dissected body. It is scarcely possible to realize how largely convention held the field in anatomical illustration up to his time, in spite of centuries of "Anatomies". Of course, as the author points out, Leonardo made mistakes, but there is nothing in the world of anatomical art to compare with the grace, poise, and accuracy of some of his sketches.

McMurrich shows how da Vinci's knowledge, based on Galenic tradition, was fostered by his master Verrochio, a name much spoken of now, when his equestrian statue of Bartholomæo Colleone is cited as an example for modern sculptors. While Leonardo escaped from the Arabian school of thought, he quotes Avicenna, and it is evident that, if we except Mondino, he had no guidance other than that

of the Arabian school to assist him in testing the accuracy of the conceptions of Galen. The author sums up his position in a single sentence, "Vesalius was undoubtedly the founder of modern anatomy. Leonardo was his forerunner, a St. John crying in the wilderness", and later he says, "If . . . the new movement in anatomy came from the artists, Leonardo may well be recognized as its originator and Vesalius as its great protagonist."

But just as when reading *The Forerunner* one was spirited away from consideration of the painter by the all-too-sketchy sidelights on Leonardo as an engineer, so here many will read with the greatest zest of him as trying out a flying machine which disappointed him sorely on many occasions; of his digressions into the fields of mechanics, dynamics, hydrostatics, and military and civil engineering; of how his work of erecting an equestrian statue of "Il Moro" was interrupted by the plague, and he did no more than produce the sketches, now one of the many treasures of Windsor Castle. In these varied capacities he came in contact with the most acute minds of his day, and it may be that to his association with Pacioli, who had become renowned by his work on the 'divine proportion', Leonardo owed the inspiration which led him to study the proportions of the human face and to reduce them to mathematical terms.

One cannot but be struck with the accuracy and agreement with modern presentation of both records and drawings, and when we compare the sketches with those which alone could serve as an example—those of Mondino's *Anathomia*—we cannot but be amazed at the extent to which Leonardo's art had advanced beyond that of his predecessors. Which brings one to the obvious inquiry: How, knowing so much and with such accurate powers of perception and illustration, did he miss the answer to the apparently simple question which obviously exercised a mind intrigued by the function of the bands traversing the ventricular cavities—a question only answered by William Harvey more than a hundred years after the death of da Vinci? In studying the circulation and the motion of the heart Leonardo allowed "theory to master observation"; that he studied its structure and the functions of its different parts very closely, and that the perfection of its mechanism excited his admiration, is evidenced by the words written under one of his illustrations of it—"wonderful instrument invented by the supreme master". Yet he accepted the Arabistic conception of three ventricles—the third being probably the left atrium; the right being regarded as part of the vena cava according to the description of Aristotle and Galen. Da Vinci drew and described the auricles, but spoke of them as ventricles. Failing to recognize the exact function of the pulmonary circulation, he accepted the Galenic hypothesis that the blood passed from the right to the left ventricle through minute pores.

The author goes on to review Leonardo's work and views upon the organs of respiration and of digestion and the nervous system, which show less inspiration than his original observations upon the heart and largely agree with the conceptions of Galen and others of his predecessors. Both in comparative anatomy and botany Leonardo's writings and drawings provide abundant evidence of his industry and powers of careful observation. In the former science he fell into the same mistake as Galen and the Salernitans in supposing that he could draw exact deductions from the anatomy of animals to that of men: much of his study was devoted to the wings of birds, on which he founded the structure of his flying machine. In botany Leonardo was a pioneer in the study of the laws of phyllotaxis (the arrangement of leaves); and to him also we owe the most interesting and far-reaching observation that the age of a tree may be determined by counting the growth-rings. All these aspects of Leonardo's activities are dealt with in a manner which will interest the amateur no less than the expert, and Professor MacMurrich concludes with an endeavour to assess his place amongst anatomists of the past. He writes that Leonardo had:—

"The faith, the vigour, bold to dwell
On doubts that drive the coward back,
And keen through wordy snares to track
Suggestion to its inmost cell."

Stepping Stones to Surgery (Anatomy Applied to Surgery). By L. BATHE RAWLING, M.B., B.Ch. (Cantab.), F.R.C.S., Surgeon to St. Bartholomew's Hospital. Demy 8vo. Pp. 228 + xvi, with 97 illustrations. 1930. London: H. K. Lewis & Co. Ltd. 12s. 6d. net.

EVEN if we cannot altogether subscribe to the accuracy of some of the observations made in this book or agree with the value and the lucidity of some of the figures, we must cordially recommend this volume in that it endeavours to encourage the study of anatomy on exactly the right lines—that is, as an adjunct to practical surgery. Thus, the author takes a case of acromegaly, and after setting forth the anatomy of the pituitary, describes the various pathological conditions which may result from diseases of that organ. This edition must, without doubt, be regarded as a venture which, if successful, is capable of considerable extension. The anatomy of the chief organs and structures of the body is illustrated by some thirty hypothetical cases, and it is to be hoped that in future editions their number will be added to with a discretion in selection equal to that of the present volume.

La Pratique chirurgicale illustrée. By VICTOR PAUCHET. Fasc. XV. Super royal 8vo. Pp. 245, with 200 illustrations drawn from nature by S. Dupret. 1930. Paris: G. Doin et Cie. Fr. 65.

THIS volume, like the preceding ones, covers a wide range of surgical subjects, all of which are fully and very ably illustrated by S. Dupret. Victor Pauchet is assisted by P. Moure on scalp grafts for replacement of eyebrows, and G. Liebault on frontal sinusitis. P. Mornard describes an interesting case of a large skin sarcoma on the inner surface of the arm. After excision this was replaced by an autoplasmic graft of skin from the side of the chest wall. The same contributor shows the result of excision of the so-called 'abdominal apron' seen in very stout women. The umbilicus is transplanted into the upper flap and makes a perfect æsthetic result.

Victor Pauchet contributes a chapter on a diaphragmatic hernia, in a boy of 13, passing through the œsophageal opening. Subserous cholecystectomy is described in detail. The separation of peritoneum from the gall-bladder is aided by air or, better, oxygen. The fixation of a drainage tube into the fossa after the gall-bladder has been removed seems to detract rather from the value of this method of removal. Chapters on gastric ulcers, perforated gastric and duodenal ulcers, gastrojejunal anastomosis after gastrectomy, and precolic gastro-enterostomy with a long loop complicated by a jejunojejunal ulcer, give great scope to the artist.

What might be termed blind drainage of the pelvis from the right iliac fossa in cases of pelvic peritonitis due to appendicitis seems a useful method in extreme cases. For tumours of the sigmoid colon an ingenious and somewhat heroic operation is described and beautifully illustrated. It consists of excision of the tumour, followed by an anastomosis of the transverse colon to the lower sigmoid colon by the intervention of a loop of small intestine (from the illustration this must be a very high loop). A similar anastomosis has been described in a previous volume (Fasc. XIII).

Inguinal hernia with ectopic testis in an adult, and excision of a seminal vesicle together with a portion of prostate, approached by the perineal route, is taken step by step, and finally the epididymis and vas deferens on the affected side are removed in cases of tuberculosis. Lastly, seventeen pages of illustrations are given showing testicular grafts from monkeys to man by Mastigues, and removal of foreign bodies from the knee-joint by Mornard. At the end of the volume is given a complete index of all the chapters in previous volumes arranged according to the part of the anatomy dealt with. This makes the series of greater value and keeps up the high standard of the work.

Surgical Pathology and Morbid Anatomy (Bowlby and Andrewes). Revised by GEOFFREY KEYNES, M.A., M.D., F.R.C.S., Assistant Surgeon, St. Bartholomew's Hospital. Eighth edition. Medium 8vo. Pp. 644 + x, with 224 illustrations. 1930. London: J. & A. Churchill. 21s. net.

THIS book has been our friend from our youth up. There is still a real need for a work of this nature written by a surgeon with pathological leanings, but there are

now perhaps few surgeons who can write such a book single-handed. That he had not been able to keep himself abreast with the progress of pathology may be one of the reasons why Sir Anthony Bowlby called in Dr. Andrewes in the preparation of the fourth edition in 1919, and it is perhaps a pity that the new editor has not similarly taken a pathologist into collaboration. for this—the eighth edition—displays on every page the evils of the endeavour to put new wine into old bottles and yet to retain the flavour of the original vintage. Matters of great pathological and clinical interest at the present time are dismissed in a few lines, and space is wasted on subjects and discussions of little value. The basis of this volume is clinical pathology, and its value and teaching can be traced back from Bowlby to Paget and ultimately to John Hunter. This ideal and certain chapters on gas gangrene and shock are well worthy of preservation, and we cannot believe that it is impossible really to revise a work of this nature.

A Treatise on Orthopædic Surgery. By ROYAL WHITMAN, M.D., M.R.C.S., F.A.C.S., Consultant to the Hospital for the Ruptured and Crippled, to St. Giles' and St. John's Guild Hospitals, etc. Ninth edition, thoroughly revised. Large 8vo. Pp. 1085 + xii, with 981 engravings. 1930. London: Henry Kimpton. 45s. net.

THIS very popular and useful text-book continues to appear in a new edition every two to three years. With each edition the book grows to a slight extent, and the present volume is no exception in this respect. From the point of view of its usefulness as a text-book, this continued increase in size is becoming a definite defect. There is now much obsolete material included—for example, no fewer than thirteen pages, with a large number of illustrations, are devoted to Abbott's method of correcting scoliosis in plaster: the account concludes with the statement that at the Hospital for the Ruptured and Crippled, the Abbott method has been discontinued in favour of correction in the upright posture. In fact, the method has been discontinued at almost every orthopædic clinic in which it has been used. Another example is the description of many methods of splinting hip disease, including all sorts of ambulatory splints which are not really in use. There is also, perhaps, a tendency to include lengthy and well-illustrated descriptions of new operations some of which are still in their trial stage, such as some of the more elaborate operations for transplanting muscles and tendons in old-standing cases of poliomyelitis. These procedures have a very limited utility, because they apply only to cases with a certain definite distribution of the paralysis. On the other hand, there are some items which would deserve greater attention. The short description of internal derangement of the knee-joint is quite inadequate by English standards, because this injury is so extremely common in all our English bone and joint clinics. Renal rickets is dismissed in three lines, and the section on late rickets is also very sketchy.

These criticisms are not intended to suggest that the book does not deserve its popularity. It still remains one of the best of orthopædic text-books, but it would benefit by a very thorough and drastic revision which aimed at a considerable reduction in its bulk.

Illustrated Primer on Fractures. Prepared by the Co-operative Committee on Fractures. Under the auspices of the Section on Surgery, General and Abdominal, and the Section on Orthopedic Surgery, in co-operation with the Department of Scientific Exhibit, of the American Medical Association. Demy 4to. Pp. 55, with 18 illustrations. 1930. Chicago: American Medical Association. \$1.00.

THIS is a book about which there will probably be two diametrically opposite views. It represents in some fifty pages a series of charts demonstrating methods of treatment of common types of fractures, with legends which, in typically trans-Atlantic language, tersely stress the chief points to be observed in carrying out treatment which is likely to meet with success. Whether such a volume is appreciated or derided, it must be admitted that the demands for its publication by a large number of the members of the American Medical Association indicates an appreciation of the medico-legal aspect of fractures and an interest in their treatment which, in the

judgement of some teachers, might with advantage be wafted over the Atlantic. The legends which adorn the pictures are much to the point. Whether there is a demand for a volume of this type in this country, and whether such a publication would produce any adequate financial return, is perhaps doubtful.

Clinique et Therapeutique chirurgicales. By GEORGE PASCALIS. *Pratique Journalière.* Medium 8vo. Pp. 290, with 80 illustrations. 1930. Paris: Gaston Doin et Cie. Fr. 50.

THE author points out in his preface that his aim is to classify the descriptions of certain symptoms occurring in a variety of surgical conditions which appear complicated to the beginner. One feels he has succeeded admirably. In this publication of 290 pages there are 80 illustrations, for which Mr. Wagner has due acknowledgement. The subjects dealt with cover a very wide field; the domains of the orthopædic surgeon, the dental surgeon, the general surgeon, and the gynæcologist are invaded.

In ruptured tendons the use of silk (No. 0) soaked in sterile vaseline is advocated, and when a graft is necessary dog's tendon preserved in alcohol. This type of heterogenous graft is also referred to in the repair of cruciate and lateral ligaments of the knee-joint. The author's abduction splint in the treatment of fracture of the upper end of the humerus is well illustrated. The chapter on old dislocations of the shoulder, after dealing with signs and symptoms, describes an operation for exposure of the joint and replacement of the head of the humerus. No way of preventing recurrent dislocations is given. Ankylosis of the hip-joint, sprains and tuberculosis of the knee-joint, and fractures of the ankle-joint, with treatment, are dealt with. Buerger's disease is gone into fully and an extremely good description of the condition is given. In the treatment of the disease peri-arterial sympathectomy gives only a temporarily good effect, and suprarenalectomy is suggested. In the chapter on acute appendicitis early operation is advocated and retrograde removal is illustrated.

In discussing the treatment of ulcers on the lesser curvature of the stomach, if medical treatment fails, local excision, or partial gastrectomy—Billroth II method—is practised. Transgastric excision is carried out in the case of the high ulcer on the posterior surface. In strangulated femoral hernia the use of local anæsthesia and division of Poupart's ligament is mentioned. Chapters are devoted to strictures of the small intestine, penetrating wounds of the abdomen, also the diagnosis and treatment of carcinoma of the rectum. With regard to the latter, 60 per cent of the cases seen are inoperable. Radium treatment is only just mentioned, and the author refers to one case in which there was an unexpected result.

In the treatment of ascites a method of injecting 6 litres of sterile water at 45° C. into the peritoneal cavity, with subsequent withdrawal half an hour later, has met with the best results. For the gynæcologist there are chapters on retroversion of the uterus, uterine fibroids, and ovarian cysts.

A good description of calculous cholecystitis is given. Cholecystectomy is the operation of choice and a subserous dissection made whenever possible. The last chapters deal with hydatid disease of the liver, anthrax, pleural fistulæ, and the treatment of fibro-adenomata of the prostate. In the latter condition the author is against the use of gauze drainage as it tends to infection and secondary hæmorrhage. He advocates Marion's suprapubic drainage tube.

The publication is a very stimulating one (of interest to the surgeon as well as the student), covering a wide range of surgical subjects, and the facts are based on the author's practical experience.

Collected Papers of the Mayo Clinic and the Mayo Foundation, Edited by Mrs. M. H. MELLISH, RICHARD M. HEWITT, M.A., M.D., and MILDRED A. FELKER, B.S. Vol. XXI. 1929. Medium 8vo. Pp. 1197 + xxiii, with 276 illustrations. 1930. Philadelphia and London: W. B. Saunders Co. 60s. net.

EACH time one opens a new volume of these papers, one is staggered by the continued expansion of the personnel of the Mayo Clinic and Foundation, and in like

proportion the output of publications from the Clinic increases. Thus in this volume of 471 papers at the disposal of the editors, 90 only are reprinted in full, 91 are abridged or abstracted, and 290 are mentioned only by title. Balfour contributes a very helpful paper on the operative treatment of ulcers of the posterior wall of the duodenum, but it is perhaps a pity that with the large amount of material at his disposal he should not have supplied some statistical support to his statement of the frequency of these lesions.

W. J. Mayo's paper on diverticulitis read before the British Medical Association at Manchester in July, 1929, presents a clear picture of the various clinical types of the disease, and the writer carries one into the medicine of the future with his interesting suggestions of the relationship of the sympathetic nervous system to the formation of acquired diverticula. In Snell's paper on recent advances in endocrinology there is much which surgeons interested in metabolism must learn to appreciate the value of and to apply. This paper contains an interesting account of hyperparathyroidism and discusses the use of parathormone. It would be well if those addicted to the universal administration of endocrine extracts would realize the warning contained in this paper with regard to the influence for harm which such substances are capable of producing when indiscriminately used.

There is only a short abstract of Waltman Walters' paper on pulmonary embolism: he shows that by the use of a regimen of which the most important feature is administration of thyroid extract—2 gr. three times a day—the incidence of embolism has during the last four and a half years been reduced to one quarter. This is a paper which every surgeon should read in full. Two papers by Lundy contrast the anæsthetic requirements for operations on the upper and those on the lower abdomen. He suggests that posterior splanchnic block has not a wide application, and makes helpful suggestions for the use of spinal anæsthesia for upper abdominal operations, though he points out that this method has its greatest field of usefulness on operations of the lower abdomen. MacCarty again urges the diagnostic value of frozen sections. It is difficult to understand why this method has not been at least accorded a trial by some of the pathologists in this country. If after a short experience such informative frozen sections can be obtained for the surgeon's guidance during the operation as those which are shown in this paper, it is surely desirable that every pathologist should have the working principles of the method at his disposal.

St. Bartholomew's Hospital Reports. Edited by Sir THOMAS HORDER, Bart., K.C.V.O., RONALD G. CANTI, WILFRED SHAW, W. LANGDON BROWN, W. GIRLING BALL, and GEOFFREY EVANS. Vol. LXIII. Demy 8vo. Pp. 277 + xxvi. Illustrated. 1930. London: John Murray. To subscribers, 15s.; to non-subscribers, £1 1s.

To write well of the life of an old master, the pupil must be in complete accord with his subject. It must be admitted that in his review of the life of Sir Anthony Bowlby, Mr. Girling Ball was fortunate in his subject; but that he fulfils his purpose well is in great part due to the fact that his task was evidently congenial. His affection and admiration for his teacher are obvious from the first page, and yet they do not obscure his judgement when he attempts to assess his position as a surgeon and to evaluate him as a man.

Bowlby's value to his hospital and medical school was considerable, but the Great War offered him an opportunity of which perhaps he was able to take greater advantage than any other living surgeon would have been, not only in co-ordinating the Medical Department with the needs of the combatant authorities, but also in conciliating the former to the demands of the doctors in their interest for the sick and wounded. It must be also borne in mind that the tact which Bowlby exercised was one of the chief factors which allowed a regular R.A.M.C. officer to find himself often in a subordinate position to a civilian with temporary rank. For the display of such qualities under all circumstances of threatening defeat and success Bowlby earned the thanks, not only of his profession, but of the whole nation. It is characteristic of the race that a very inadequate appreciation of the services he

rendered was granted to him in his lifetime when we compare it with the honours and gratuities bestowed upon members of the fighting forces for what was certainly a much less successful record in positions of vastly less difficulty.

Mr. T. A. Lodge provides an interesting record of the new surgical block which, though written by an architect, will be easily comprehended and of great interest to any surgeon who is taking a share in a similar enterprise.

Dr. Dicks discusses the clinical value of Lyon's method of transduodenal biliary drainage. Mr. Raven contributes a careful analysis of eighty cases of perforated duodenal and gastric ulcers, and Mr. Corbett endeavours to assess the period of disability in ambulatory fractures from a review of some 250 cases treated in the Fracture Clinic.

The Dawn and Epic of Neurology and Surgery. By Sir CHARLES BALLANCE, K.C.M.G., C.B., LL.D., F.R.C.S. 8vo. Pp. 40. Illustrated. 1930. Glasgow: Jackson Wylie & Co.

SIR CHARLES BALLANCE, in this the Macewen Memorial Lecture for 1930, traces neurology from the time of prehistoric trephining to the present day, when it has culminated in the work of Dr. Harvey Cushing of Boston, Massachusetts, and of Sir Percy Sargent in London. Surgical neurology—if such a term is permissible—was empirical and rarely practised until Ferrier, Victor Horsley, Sir William Macewen, and Ballance himself, to speak only of the pioneers in Great Britain, based it on experiment and thus made it scientific. The thesis sustained is that operative neurology needs the devotion of a lifetime and should only be undertaken by those who are specially drawn to the subject. The lecture is illustrated with portraits of Macewen, Pasteur, Lister, Hughlings Jackson, David Ferrier, John Hunter, and Harvey Cushing. It is published as No. XVI of the Glasgow University Publications, and well repays perusal.

Shorter Convalescence. By Lieut.-Colonel JAMES K. McCONNEL, D.S.O., M.C., Member of the Chartered Society of Massage and Medical Gymnastics; Bio-physical Assistant (working at St. Thomas's Hospital). With a Foreword by Sir ROBERT JONES, Bart., K.B.E., C.B., F.R.C.S. Crown 8vo. Pp. 132 + xi. Illustrated. 1930. London: William Heinemann (Medical Books) Ltd. 5s. net.

COLONEL McCONNEL has written a little volume which will well repay study by the operating surgeon, particularly the surgeon who carries out many routine abdominal operations. The idea is to suggest methods whereby the period of flabbiness following an illness or an operation may be curtailed. He points out the common but erroneous idea that exercises necessarily involve extensive movements with considerable muscular effort. What is needed in convalescence is, rather, small exercises introducing co-ordination and control of muscular activity. Such exercises can be quite well given in bed, and the automatic co-ordination of movements involved in standing and walking can be taught before the patient gets up.

The book is small, but not easily read; although the descriptions are somewhat involved, they are so arranged as to add interest to the subject, and the surgical reader should not be put off if he finds a little difficulty in following the author's ideas.

Surgery of the Lung and Pleura. By H. MORRISTON DAVIES, M.A., M.D., M.Ch. (Cantab.), F.R.C.S., Medical Superintendent, Vale of Clwyd Sanatorium, etc. Demy 8vo. Pp. 355 + xvi, with 104 illustrations. 1930. London: Oxford University Press. 28s. net.

THIS book on intrathoracic surgery is based upon a former volume by the same author published in 1919, but, as the author states in the preface, owing to the enormous advance in intrathoracic surgery, apart from the chapter on anatomy, which has been revised, the whole book has been rewritten and the greater number of the illustrations are new.

The chapter upon physiological considerations largely embodies the researches and supports the conclusions of Graham, whose work has revolutionized the treatment

of certain types of purulent pleural effusions. The section upon the treatment of chronic empyema, a most difficult condition to cure, is somewhat disappointing, as one would have expected the author to have given more definite indications for the different operations which are mentioned in the text. The chapter on pulmonary tuberculosis, which includes a section upon treatment by artificial pneumothorax, is full of information, and can be recommended as a non-controversial account of the present position of surgery in this disease. The succeeding review of phrenic evulsion, thoracoplasty, and extrapleural pneumolysis contains an account of the comparative value of artificial pneumothorax and thoracoplasty. Each can be shown to have certain advantages. In the operation itself the author recommends the use of chloroform combined with local novocain as the means of anaesthesia, and makes the statement that "the after-condition is also more satisfactory than that following gas and oxygen", a statement which is open to considerable doubt.

Bronchiectasis and pulmonary abscess are dealt with upon accepted lines, but the value of the administration of arsenical compounds, especially in the latter condition, would appear worthy of mention. There is an excellent chapter on hydatid cysts, but that on primary tumours of the lung and mediastinal dermoids is short and contains very little of the recent work on diagnosis and treatment of these conditions.

The small amount of criticism necessary shows that the book is fully representative of the generally accepted teaching of the surgery of the lung and pleura at the present day, and can be thoroughly recommended.

Cancer of the Lung and other Intrathoracic Tumours. By MAURICE DAVIDSON M.A., M.D., B.Ch. (Oxon.), F.R.C.P. (Lond.), Physician to the Brompton Hospital for Diseases of the Chest, etc. With a Foreword by ARTHUR J. HALL, M.A., M.D., D.Sc., F.R.C.P. (Lond.), Professor of Medicine, University of Sheffield. Super royal 8vo. Pp. 173 + x, with 62 illustrations. 1930. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

THE increasing interest and the numerous papers in the medical press on malignant disease in the chest have long necessitated the production of a monograph upon this important subject. This want has been filled by the volume under review. The book contains chapters upon the history and statistical aspects, morbid anatomy and histology, etiology, clinical and radiological aspects, diagnosis, and treatment. It has, in addition, a chapter upon non-malignant intrathoracic tumours.

From the figures quoted it would appear that there has been a definite absolute increase in the incidence of malignant disease of the lung during the last twenty years, but although various predisposing factors such as influenza, tobacco, war gas, petrol fumes, and road tar have been held to account for the increase, no observations have been made so far which would justify conclusions on this point. The association between tuberculosis and cancer is pointed out as incidental rather than one of cause and effect, a view which would appear to be confirmed by the statistics of the autopsies at Brompton Hospital, in which only 6.5 per cent of the cancer cases had definite associated tuberculous disease. The clinical section is made more interesting by the inclusion of the notes of a series of twenty-two cases.

The greater portion of the recent improvement in the diagnosis of intrathoracic disease is due to the radiologist, and this aspect is fully discussed, and well illustrated by some excellent reproductions of skiagrams of pulmonary growths of various types.

The chapter on treatment is of considerable interest, as it indicates the view of the more progressive physicians towards surgical measures in the chest. The author states that the "advance in our operative technique in the last few years has been such that it can now be said with truth that in expert hands the operative risks of intrathoracic surgery are no greater than those of abdominal surgery, and that manipulations within the chest carry with them no more anxiety than any recognized major operation within the peritoneal cavity; in some cases it may be said to be less."

This volume can be recommended to all those interested in chest disease as an excellent short review of the present position of our knowledge in regard to malignant disease in the chest.

Die Knochenbrüche und ihre Behandlung. Ein Lehrbuch für Studierende und Ärzte. By Prof. Dr. HERMANN MATTI (Bern). Second edition. Royal 8vo. Pp. 938 + xvi, with 1000 illustrations. 1931. Berlin: Julius Springer. Paper covers, RM. 86; bound, RM. 89.60.

THIS is a very full and complete account of the subject of fractures and their treatment by one of the Berne school, who has dedicated his work to Kocher. The great size of the work is accounted for by the fact that it includes the subjects of cerebral and spinal complications of the fractures of the skull and vertebral column; and also by the lavish wealth of the illustrations.

The first part (237 pp.) is occupied by a discussion of general principles, anatomy, pathology, and treatment, and is on the whole well balanced and inclusive, although the sections on operative methods are much less complete than those on traction methods of treatment. The second part (167 pp.) is concerned with fractures of the skull and their cerebral complications. The different phases of cerebral symptoms are described and discussed with great care and detail, and both diagnosis and treatment are fully dealt with. This section includes a good account of gunshot injuries of the skull and brain.

The next section (63 pp.) deals with fractures of the spine, and, like all other parts of the work, is very copiously illustrated. The different types of lesion and the symptoms produced are very fully given, but the latent type of disease described by Kümmell does not seem to have a distinct enough recognition, and the section on treatment is disappointing as giving so scanty a reference to methods and indications for operative fixation.

The remainder of the volume gives a systematic description of fractures of individual bones, and in its wealth of illustration affords a most useful book of teaching and reference. As an exposition of modern traction methods of treatment this book is very complete and convincing, but as a comprehensive account of modern fracture treatment, and especially from the point of view of open operations, it is distinctly disappointing. The great charm and value of the book is the number of illustrations and the beautiful way in which they are reproduced.

La Pratique du Pneumothorax thérapeutique et de la Collapsothérapie chirurgicale. By F. DUMAREST and P. BRETTE. Medium 8vo. Pp. 409 + xiii. Illustrated. 1929. Paris: Masson et Cie. Fr. 50.

THIS volume is an enlarged revision, with the addition of a chapter on surgical collapse, of two previous editions of *La Pratique du Pneumothorax thérapeutique*. An unnecessarily long account is given of various forms of apparatus, needles, etc., for the production of pneumothorax. The authors regard pleural shock as a generic term for the two conditions of pleural reflex and gas embolism. Its avoidance can be largely ensured by careful attention to operative details, although the value of adequate local anaesthesia does not appear to be sufficiently stressed in this connection. The further treatment of pulmonary tuberculosis by artificial pneumothorax is fully discussed, and the value of the treatment is considered to be due to its allowing spontaneous contraction of the lung rather than to active compression.

The pleural group of complications are common, and the figures quoted show that the frequency of pleural effusions of all types varies with the different authors quoted, from 52 per cent (Rist and Naveau) to 100 per cent (Matson, Matson and Bisailon). Unquestionably the routine use of screen examination at frequent intervals in all cases accounts for the high figures of the latter authorities, evanescent effusions, of no moment in many cases, being included. The more striking figures are those of Dumarest, in which between 16 and 18 per cent of purulent effusions supervene. As one would expect under these circumstances, the treatment of these effusions is fully described.

Oleothorax is advised in contracting pneumothorax, for the obliteration of pleuropulmonary fistula, and in certain cases of purulent pleural effusions, either simple or secondarily infected, all conditions which are otherwise often difficult to control, but the value of oleothorax in all except contracting pneumothorax is debatable.

A short chapter is devoted to thoracoscopy and the canterization of adhesions, but owing to the inexperience of the authors with the method of treatment, it is neither recommended nor condemned. The concluding chapter on surgical collapse contains a description of the operations of phrenicotomy and thoracoplasty, with their indications, complications, and results.

This book, taken as a whole, can be relied upon as an adequate presentation of treatment by artificial pneumothorax, although that portion devoted to surgical collapse falls somewhat below the general standard of the remainder of the volume.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Souvenir—Cinchona Tercentenary Celebration and Exhibition at the Wellcome Historical Medical Museum. By HENRY S. WELLCOME, LL.D., F.S.A., and L. W. G. MALCOLM, M.Sc. (Cantab.), F.R.S.E. 9 $\frac{3}{4}$ " \times 6 $\frac{3}{4}$ " Pp. 115. Illustrated. 1930. London: The Wellcome Foundation Ltd.

William Stewart Halsted: Surgeon. By W. G. MACCALLUM. Introduction by Dr. W. H. WELCH. Large post 8vo. Pp. 241 + xvii. Illustrated. 1930. Baltimore: The Johns Hopkins Press. London: Oxford University Press. 12s. 6d. net.

Radium Therapy: Principles and Practice. By G. E. BIRKETT, M.C., B.A. (Cantab.), M.R.C.S., Hon. Radiologist, Manchester and District Radium Institute and Christie Hospital for Cancer. Royal 8vo. Pp. 186 + x, with 6 colour and 13 half-tone plates and 52 illustrations in the text. 1931. London: Cassell & Co. Ltd. 17s. 6d. net.

Pye's Surgical Handicraft. Edited by H. W. CARSON, F.R.C.S., Late Senior Surgeon, Prince of Wales's General Hospital, Tottenham; Lecturer on Abdominal Surgery, North-east London Post-graduate College. Tenth edition, fully revised. Medium 8vo. Pp. 641 + xviii, with 22 plates and 343 illustrations in the text. 1931. Bristol: John Wright & Sons Ltd. 21s. net.

Sir D'Arcy Power: Selected Writings, 1877-1930. Medium 8vo. Pp. 368 + x. Illustrated. 1931. Oxford: The Clarendon Press. 25s. net.

Cancer and Scientific Research. By BARBARA HOLMES, Ph.D. (Cantab.), Biochemical Laboratory, Cambridge. With a Preface by Professor Sir F. G. HOPKINS, President of the Royal Society. Crown 8vo. Pp. 160. 1931. London: The Sheldon Press. 3s. 6d. net.

Guy's Hospital Reports. Edited by ARTHUR F. HURST, M.D. Vol. LXXX (Vol. X. Fourth Series). October, 1930. Royal 8vo. Pp. 379-500. Illustrated. 1930. London: The Lancet Ltd. Single numbers, 12s. 6d. net; annual subscription, £2 2s. net.

Spezielle chirurgische Diagnostik für Studierende und Ärzte. By Dr. F. DE QUERVAIN (Bern). Ninth edition, fully revised. Super royal 8vo. Pp. 916 + xvi, with 833 illustrations in the text and 6 plates. 1931. Leipzig: F. C. W. Vogel. Paper covers, RM. 75; bound, RM. 78.60.

Die äusseren Abdominal-Hernien. By Dr. ERICH BUMM, Assistent an der Chirurgischen Universitätsklinik, Berlin. Imperial 8vo. Pp. 331 + viii, with 235 illustrations. 1931. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 40; bound, RM. 42.50.

690 THE BRITISH JOURNAL OF SURGERY

- Modern Surgery: General and Operative.** By JOHN CHALMERS DA COSTA, M.A., LL.D., F.A.C.S., SAMUEL D. GROSS, Professor of Surgery, Jefferson Medical College, Philadelphia, etc., assisted by BENJAMIN LIPSHUTZ, M.D., F.A.C.S., Surgeon to the Mt. Sinai Hospital, Associate in Neuro-anatomy, Jefferson Medical College, etc. Tenth edition, revised and reset. Royal 8vo. Pp. 1404, with 1050 illustrations. 1931. Philadelphia and London: W. B. Saunders Co. 45s. net.
- Diseases of the Tongue.** By WALTER G. SPENCER, M.S., F.R.C.S., Consulting Surgeon, Westminster Hospital; and STANFORD CADE, F.R.C.S., Assistant Surgeon, Westminster Hospital. Being the third edition of Butlin's *Diseases of the Tongue*. Demy 8vo. Pp. 561 + xvi, with 123 illustrations in the text and 20 coloured plates. 1931. London: H. K. Lewis & Co. Ltd. 35s. net.
- Clinical Examination and Surgical Diagnosis.** By FÉLIX LEJARS, Professeur de Clinique chirurgicale à la Faculté de Médecine de Paris. Translated by HELEN C. SCOTT, M.R.C.S., L.R.C.P. Large 8vo. Pp. 872 + xi, with 1094 illustrations. 1931. London: Johnathan Cape Ltd. 50s. net.
- Die Chirurgie des vegetativen Nervensystems.** By ERICH HESSE (Leningrad). Large 8vo. Pp. 475, with 133 illustrations in the text and 14 coloured plates. 1930. Moscow and Leningrad: Staatsverlag.
- Annals of Roentgenology: a Series of Monographic Atlases.** Edited by JAMES T. CASE, M.D., Professor of Roentgenology, Northwestern University Medical College. Chicago. Volume XII. The Chest in Children. By E. GORDON STOLOFF, M.D. Crown 4to. Pp. 432 + xxx, with 401 illustrations. 1931. New York: Paul B. Hoeber Inc. \$15.00.
- Clio Medica Series.** A series of Primers in the History of Medicine. Editor: E. B. KRUMBHAR, M.D. Fcap 8vo. 1930 and 1931. New York: Paul B. Hoeber Inc. \$1.50 per volume.
- The Beginnings: Egypt and Syria.** By WARREN R. DAWSON, F.R.S.E., Fellow of the Royal Society of Medicine, etc. Pp. 86 + x.
- Medicine in the British Isles.** By SIR D'ARCY POWER, K.B.E., F.R.C.S., Hon. Librarian at the Royal College of Surgeons of England, etc. Pp. 84 + x. Illustrated.
- Anatomy.** By GEORGE W. CORNER, M.D., Professor of Anatomy in the University of Rochester. Pp. 82 + xvii. Illustrated.
- Internal Medicine.** By SIR HUMPHRY ROLLESTON, Bart., G.C.V.O., K.C.B., M.D., Hon. D.Sc., D.C.L., LL.D., [Regius Professor of Physic in the University of Cambridge. Pp. 92 + x.
- Physiology.** By JOHN F. FULTON, M.D., Stirling Professor of Physiology, Yale University. Pp. 141 + xvi. Illustrated.
- Chirurgie esthétique pure (Technique et Résultats).** By RAYMOND PASSOT, Ancien Interne des Hôpitaux de Paris. Collection des Actualités de Médecine pratique edited by Dr. R. J. WIESSENBAUGH, Médecin des Hôpitaux de Paris. Crown 8vo. Pp. 283 + xx, with 80 illustrations in the text and 8 plates. 1931. Paris: G. Doin et Cie. Fr. 45.
- Cancer and Race: a Study of the Incidence of Cancer among Jews.** By MAURICE SORSBY, M.D., F.R.C.S.E., Hon. Assistant Surgeon, Ear, Nose, and Throat Department, London Jewish Hospital, etc. With a Preface by Lieut.-Col. F. E. FREMANTLE, M.A., M.D., M.Ch., F.R.C.P., F.R.C.S., D.P.H., M.P., Consulting County Medical Officer of Health for Herts. Conducted under the auspices of the Jewish Health Organization of Great Britain. Demy 8vo. Pp. 120 + xvi. 1931. London: John Bale, Sons & Danielsson, Ltd. 7s. 6d. net.
- Fractures and their Complications.** By GEORGE EWART WILSON, M.B. (Tor.), F.R.C.S., F.A.C.S., Surgeon-in-Chief, St. Michael's Hospital, Toronto, etc. Medium 8vo. Pp. 415 + viii, with 306 illustrations. 1931. London: Ballière, Tindall & Cox. 35s. net.
- Chirurgische Krankengymnastik.** By DR. KARL GEBHARDT, Assistant der Chirurgischen Universitäts-Klinik, München (Geheimrat Lexer), und Oberarzt der Sportabteilung. Imperial 8vo. Pp. 46, with 37 illustrations. 1931. Leipzig: Johann Ambrosius Barth. RM. 3.8.

INDEX TO VOLUME XVIII

	PAGE		PAGE
ABDOMINAL emergency, purpura as an acute (<i>see</i> Purpura)		Anaerobic infections, gall-bladder disease and - - - - -	78
Acetabulum, tuberculosis of (<i>see</i> Tuberculosis of Hip)		Anæsthesia, intravenous local - - -	641
ACKMAN, F. D., and RHEA, LAWRENCE J.: Non-parasitic cysts of the liver: their clinical and pathological aspects - - - -	618	— — — application of experimental results to man - - -	646
Adamantinoma of tibia, primary - - -	415	— — — effect on blood-pressure and respiration - - -	644
— — — clinical notes - - -	415	— — — experimental investigations - -	643
— — — comment on case report - - -	420	— — — historical note - - -	641
— — — pathological examination - -	417	— — — indications - - -	646
Adenocarcinoma of bladder (<i>see also</i> Bladder: Urachus) - - -	422	— — — technique - - -	641
— near umbilicus - - -	461	— — — type of anæsthesia - - -	642
Adenomata of salivary glands (<i>see</i> Salivary Glands, Mixed Tumours of)		— local, in treatment of fractures (<i>see</i> Fractures)	
— urachal (<i>see</i> Urachus)		Anaphylaxis, hydatid - - - - -	278
Adhesions, mechanical ileus due to, after gastrojejunostomy - - -	156	Anatomical result of peri-arterial sympathectomy - - -	215
Adhesive strapping, traction by, in tuberculosis of hip - - -	59	Aneurysms, arteriovenous, competency of valves of vein in - - -	53
Adson's operation on Gasserian ganglion	147	— intracranial - - - - -	150
Aerocele, intracranial, with frontal sinus osteoma - - -	572	Angioma of bladder (<i>see</i> Bladder, Hæmangioma of)	
— — traumatic - - -	655	— kidney (<i>see</i> Kidney, Hæmangioma of)	
'Aerocolic', volvulus of stomach and - - -	105, 106	— retroperitoneal - - - - -	523
Africans, hernia in - - -	16	Angular deformity of spine, fatal cardiac failure with - - -	326
Age changes in intervertebral discs	361, 370	Ankle, Charcot's disease of - - -	113
— incidence of cysts of bones of hand and foot - - -	30	— fracture-dislocation of, local anæsthesia in treatment - - -	551, 552
— — frontal-sinus osteomata - - -	569	— injuries of, intravenous anæsthesia for - - -	646
— — urachal tumours - - -	443	— — synovial folds in - - -	637
— — volvulus of stomach - - -	105	Anoxæmia in cardiac surgery - - -	273
AITKEN, R. Y.: Cyst of the ileum - -	521	Aorta, traumatic rupture of - - -	664
Albee's route to hip-joint - - -	49	'Apparatus major' operation for stone	3
— saw in cranial surgery - - -	225	— 'minor' operation for stone - - -	1
Albocasse (Peter de), physician in Henry IV's reign - - -	492	Appendicectomy, gas gangrene following - - -	43
ALCOCK, ARNOLD: A case of intracranial aerocele or traumatic pneumoœranium - - -	655	Appendix, strangulation of - - -	531
Alkaptonuria, spinal lesions in - -	370	Arderne, John of - - -	492
ALLEN, I. M., WAKELEY, CECIL P. G., WORSTER-DROUGHT, C., and DICKSON, W. E. CARNEGIE: Endothelioma of the cauda equina. Report of a case with removal of tumour and recovery - -	6	ARMITAGE, GEORGE: Intestinal obstruction complicating posterior gastrojejunostomy: a case of internal strangulation of small intestine by the afferent limb - -	154
Altoftes (William), surgeon to Henry VII	498	— — Osteoma of the frontal sinus: with particular reference to its intracranial complications, and with the report of a case - -	565
Ambulatory splint for tuberculosis of hip	63	ARMOUR, J. C., et al.: Studies on high intestinal obstruction: the administration of saline and other substances by enterostomy below the site of obstruction - -	467
Ametox for pigmentation due to bismuth absorption - - -	330	Arsdale's (Van) saw for cranial surgery	224
Ampulla of Vater, cancer of - - -	85, 86, 87		
Amputations, intravenous anæsthesia for	646		

	PAGE		PAGE
Arteries, changes in, after peri-arterial sympathectomy - - -	215	Bismuth absorption after use of B.I.P.P., pigmentation from - - -	329
Arteriosclerosis, relationship to degeneration of intervertebral discs -	372	Bladder, adenomata occurring in trigone and neighbourhood - -	437
Arteriovenous aneurysm, competency of valves of vein in - - -	53	— amount to be removed in operation for urachal tumours - - -	445
Arthritis, neuropathic, pathology of -	113	— diverticulum of, angioma in - -	209
— of spine, intervertebral discs in -	366, 374	— ectopic, tumours arising in upper part of - - - - -	433
Arundel (John), surgeon in Henry VI's reign - - - - -	495	— hamangioma of - - - - -	205
Astrocytomata (<i>see</i> Intracranial Tumours)		— case reports - - - - -	205
Asymmetry, facial, idiopathic - - -	202	— commentary - - - - -	212
Atresia of ileum, congenital - - -	333	— review of the literature - - -	208
		— symptomatology - - - - -	212
		— treatment - - - - -	213
		— vesical varices and - - - - -	212
<i>BACILLUS welchii</i> infections, gall-bladder disease and - - -	80, 81	— 'mixed tumours' of - - - - -	433, 437, 446
— rôle in intestinal obstruction -	467	— vault, adenocarcinomata of (<i>see also</i> Urachus, Tumours of) - - -	422
Bacteriological study of fifty cases of cholecystectomy, with special reference to anaerobic infections -	78	BLAIR, D. M., DUFF, D., and BINGHAM, J. A.: The anatomical result of peri-arterial sympathectomy -	215
Bacteriology of gas gangrene - - -	47	Bleeding from portal vein during operation for hydatid of liver - - -	585
BAILEY, HAMILTON: Purpura as an acute abdominal emergency - -	234	— in wall of small gut in purpura -	234
BAKER, ALFREDA H., and HAWKSLEY, L. M.: A case of primary adamantinoma of tibia - - - - -	415	Blood chemistry in intestinal obstruction — effect of experimental mitral obstruction on - - - - -	262, 265
Baldwin, physician to Edward the Confessor - - - - -	480	— extravasation of, subserosal, in purpura - - - - -	234, 238
Barker's approach to hip-joint - - -	49	— infection, staphylococcal, osteomyelitis of spine due to - - - -	400, 401
Beauveys (Simon and Philip de), surgeons to Edward I - - - - -	485	Blood-pressure, effect of intravenous novocain on - - - - -	614
Bed used in Pugh's method in tuberculosis of hip - - - - -	60	Blood-supply of sternomastoid muscle	197
BEGG, R. CAMPBELL: The colloid carcinomata of the bladder vault arising from the epithelium of the urachal canal: with a critical survey of the tumours of the urachus - - - - -	422	Bone absorption in Chareot's joints -	121
Bekbank (Thomas), groom-surgeon to Henry VI - - - - -	496	— aspects of pathology of hypertrophic Chareot's joints - - -	113
Bellmont (Ralph de), physician to Henry II - - - - -	480	— changes associated with congenital torticollis - - - - -	199
Belwell (Michael), surgeon under Henry VI - - - - -	495	— growth in embryo - - - - -	118
BERRY, ALLAN J.: Chronic subluxation of the distal radio-ulnar articulation - - - - -	526	Bone-flaps in osteoplastic craniotomy (<i>see</i> Craniotomy)	
Besace (Ramulphus), physician to Richard I - - - - -	480	Bone-grafting for cysts of long bones of hand and feet - - - - -	36
Bier's (A.) method of intravenous local anaesthesia - - - - -	641	— tuberculosis of hip - - - - -	62
Bile-ducts, rupture of hepatic cyst into Biliary calculi, bacteriology of - -	287	Bones of hand and foot, cysts of (<i>see</i> Cysts)	
— diagnosis from hydatid disease involving ducts - - - - -	288	Brachial plexus, pressure of cervical rib on, vascular symptoms from -	560, 562
— fistula, external, transplanted into duodenum - - - - -	581	Bradwardyn (William), surgeon to Richard II - - - - -	492, 493
BINGHAM, J. A., DUFF, D., and BLAIR, D. M.: The anatomical result of peri-arterial sympathectomy -	215	Brain, cysts of, secondary - - - - -	285
B.I.P.P., pigmentation due to use of -	329	— effect of radium on normal cerebral tissue - - - - -	622
Birth injury, congenital torticollis and sternomastoid tumour and - - -	188, 198	— tumours of (<i>see</i> Intracranial Tumours)	
		Braun's method of intra-arterial local anaesthesia - - - - -	641
		Breast, cancer of, rodent ulcer following prolonged X-radiation of - -	96
		Brightman (Richard), surgeon under Edward IV - - - - -	496
		Broncho-hepatic fistula - - - - -	289
		Bronchus, rupture of pulmonary cyst into - - - - -	290

	PAGE		PAGE
BROWN, T. G., et al.: Studies on high intestinal obstruction: the administration of saline and other substances by enterostomy below the site of obstruction -	467	Cats, experimental mitral obstruction in -	263
Brown-Séquard syndrome, partial, with spinal tumour -	670, 672	— experiments with intravenous novocain on -	643
BUCHANAN, JAMES: Volvulus of the stomach -	99	Cauda equina, endothelioma of: report of case with removal of tumour and recovery -	6
Bygone Operations in Surgery:—		Cauterization with carbolic acid in miniature bone cysts -	36
Cutting for the stone: Apparatus minor and apparatus major operations -	1	Cavernous hamangioma of kidney (<i>see</i> Kidney)	
Cutting for the stone: Cheselden's operation -	353	Cerebral and cerebellar tumours (<i>see</i> Intracranial Tumours)	
Cutting for the stone: Frère Jacques' operation -	185	— cysts, secondary -	285
An historical lithotomy: Mr. Samuel Pepys -	541	— tissue, normal, effect of radium on -	622
		Cerebrospinal fistulae closed by fascial implants -	577
CADE, STANFORD, and SARGENT, SIR PERCY: The treatment of gliomata and pituitary tumours with radium -	501	— fluid, changes in endothelioma of cauda equina -	7, 13
Calcification of intervertebral discs -	365	— — effect of irradiation on -	507
Calcinosis intervertebralis -	365, 371	— rhinorrhea with frontal-sinus osteoma -	572
Calculi, biliary, bacteriology of -	78	Cervical rib, vascular complications of -	557
— — diagnosis from hydatid disease involving ducts -	288	Chabo (Anthony), surgeon to Henry VIII -	498
— renal, with squamous carcinoma in a hydronephrotic kidney -	590	Chambers (Richard), surgeon under Edward IV -	497
— urethral (<i>see</i> Cutting for the Stone)		Charcot's joints, hypertrophic, some aspects of pathology of -	113
Cancer (<i>see also</i> under various organs)		Cheselden's operation for the stone -	353
— ampullary -	85, 86, 87	Chlorides administered by enterostomy in high intestinal obstruction -	467
— of breast, rodent ulcer following prolonged X-radiation of -	96	Cholecystitis, bacteriology of -	78
— with calculi, in hydronephrotic kidney -	590	Cholelithiasis, bacteriology of -	78
— intestine, incidence -	85	— diagnosis from hydatid disease involving bile-ducts -	288
— pylorus, duodenum involved in -	84	Chondromatous cysts of long bones of hand and foot (<i>see</i> Cysts)	
— vertebrae -	366	Choroid plexus, effect of irradiation on -	507, 632
Carbolic acid, use in miniature bone cysts -	36	Circular saws for craniotomy -	223, 225
Carcinoma (<i>see</i> Cancer)		Circulatory system of sternomastoid muscle -	189, 197
Cardiac cysts -	285	Clarnbold, physician to Henry I -	480
— failure, fatal, with spinal deformity -	326	Clavicular exostosis associated with congenital wry-neck -	199
— surgery (<i>see</i> Mitral Obstruction)		Cocaine, experimental work on injections of -	646
Cardioscope for use in cardiac surgery -	268	Cole (Simon), surgeon under Edward IV -	497
Caries of spine -	401, 402	Collard (Thomas), surgeon under Edward IV -	496
— — fatal cardiac failure with -	326	Colles's fracture, local anæsthesia in treatment -	546, 547, 549
— — intervertebral discs in -	367	Colloid adenocarcinomata of bladder vault (<i>see</i> Bladder Urachus)	
Carliolo (Patrick de), physician under Henry III -	483	Colnet (Nichol), physician to Henry V -	494
Carpal joint, lesions of, intravenous anæsthesia for -	646	Colon, gas-distension of, volvulus of stomach and -	105, 106
— — synovial folds in -	636	— involved in volvulus of stomach -	103, 104
— scaphoid, local anæsthesia in treatment -	551	Colostomy, gas gangrene following -	43
CARSON, H. W.: Acute osteomyelitis of the spine -	400	Columbia-paste skull-cap for radium treatment of brain tumours -	507, 632
Cartilage formation by tendon-sheath tumours -	608	COLWELL, H. A., CREED, E. FF., and EDWARDS, HAROLD: Rodent ulcer as the sequel to prolonged X-radiation -	96
— hyaline, case of osteitis fibrosa with formation of -	409	Compression fracture of vertebrae -	366

	PAGE		PAGE
Congenital absence of radius with non-rotation of intestine - - -	166	Cysts of long bones of hand and foot, osteitis fibrosa and - - -	20, 31, 35
— atresia of small intestine - - -	333	— — — — pathogenesis - - -	31
— cysts of liver (<i>see</i> Liver, Non-parasitic Cysts of)		— — — — skeletal site - - -	30
— stenosis of duodenum - - -	331	— — — — summary - - -	36
— torticollis (<i>see</i> Torticollis, Congenital)		— — — — symptomatology - - -	29
— unilateral polycystic kidney in an infant - - - - -	162	— — — — treatment - - -	35
CONNELL, W. K. : Hernia in Africans -	16	— — — — X-ray signs - - -	30
COOMBS, CAREY F. : Fatal cardiac failure occurring in persons with angular deformity of spine -	326	— retroperitoneal, case of large retroperitoneal lymphangioma - -	523
Cranial nerves, relation to Gasserian ganglion - - - - -	128	— unilateral polycystic disease of kidney in an infant - - -	162
Craniotomy, osteoplastic - - -	221		
— — circular saws for - - - 223,	225	DAVID, surgeon to Henry III - -	482
— — Gigli saws for - - -	224	Decompression prior to radium treatment of brain tumours -	503
— — historical - - -	222	Dermoid cyst simulating gastric ulcer -	339
— — introduction - - -	221	— thoracic, case report - - -	666
— — mechanical craniotomes for -	224	DEW, HAROLD : Some complications of hydatid disease - - -	275
— — methods of performing - - -	222	Diaphragmatic hernia, traumatic -	170
— — problem of improving Gigli-saw technique - - -	225	Diathermy, per-urethral, in vesical angiomata - - -	213
— — 'skull plough' for use in - -	225	DICKSON, W. E. CARNEGIE, WAKELEY, CECIL P. G., WORSTER-DROUGHT, C., and ALLEN, I. M. : Endothelioma of the cauda equina. Report of a case with removal of tumour and recovery - - -	6
— — — — advantages - - -	232	Digits, cysts of (<i>see</i> Cysts)	
— — — — case report - - -	230	— fractures of, local anaesthesia in -	553
— — technique of cutting and turning down bone-flaps from skull -	227	— gangrene of, due to cervical rib -	557
CREED, E. FF., EDWARDS, HAROLD, and COLWELL, H. A. : Rodent ulcer as the sequel to prolonged X-radiation - - - - -	96	Dilatation of veins, competency of valves in - - - - -	53
Curettage of miniature bone cysts -	36	Dislocations of carpal and tarsal joints, intravenous anaesthesia for -	646
'Cutting on the gripe' - - -	1	Diverticula of urachal canal - - -	451
Cutting for the stone : apparatus minor and apparatus major operations -	1	Diverticulum of bladder, angioma in -	209
— — Cheselden's operation - - -	353	Dogs, experimental mitral obstruction in - - - - -	263, 271
— — Frère Jacques' operation - - -	185	— muscle changes after venous ligation in - - - - -	193
— — an historical lithotomy : Mr. Samuel Pepys - - - - -	541	Doyen's saw for cranial surgery - -	223
Cyst, dermoid, simulating gastric ulcer -	339	DUFF, D., BLAIR, D. M., and BINGHAM, J. A. : The anatomical result of peri-arterial sympathectomy -	215
— hydatid (<i>see also</i> Hydatid Disease)		DUNLOP, D. M. et al. : Studies on high intestinal obstruction : the administration of saline and other substances by enterostomy below the site of obstruction - - -	467
— of liver. (<i>see</i> Liver)		Duodenal ulcer (<i>see</i> Gastric and Duodenal Ulcer)	
— lung, (<i>see</i> Lung)		Duodenum, cancer of - - - - -	84
— of neck - - - - -	338	— — case report - - - - -	88
— of ileum - - - - -	521	— — distribution - - - - -	85
— intestinal obstruction due to - -	168	— — etiology - - - - -	85
Cystitis cystica - - - - -	436	— — incidence - - - - -	84
Cystoscopic appearances of colloid cancer of urachus - - - - -	439	— — relationship to ulcer - - -	86
Cysts, formation of, in urachus -	450	— — symptomatology - - - - -	86
— of liver, non-parasitic (<i>see</i> Liver)		— — treatment - - - - -	87, 90
— — long bones of hand and foot -	20	— congenital stenosis of - - -	331
— — — — age and sex incidence -	30	— external biliary fistula transplanted into - - - - -	581
— — — — analysis of cases - - -	29		
— — — — case reports - - -	21		
— — — — chondromatous type - - -	31, 35, 36		
— — — — clinical signs - - -	29		
— — — — diagnosis - - -	35		
— — — — indications for operation - - -	36		
— — — — introduction - - -	20		
— — — — operative technique - - -	36		

	PAGE		PAGE
E CHINOCOCCOSIS, secondary (see Hydatid Disease)		Fibro-adenoma at or near umbilicus	461
ECKHOFF, NILS L.: Gas gangrene in civil surgery	38	— of urachus (see also Urachus)	433, 458, 461
Ectopic bladders, tumours arising in upper part of	433	Fibroma and fibromyoma of urachus (see also Urachus, Tumours of)	433
EDWARDS, HAROLD, CREED, E. W., and COLWELL, H. A.: Rodent ulcer as the sequel to prolonged X-radiation	96	— of mesentery	658
Elbow-joint, operation for complete exposure of	303	— partially ossified, of stomach wall	660
— synovial folds in	637	Fifth-nerve area, herpes in	142
Electrocardiographic changes in experimental cardiac obstruction	263, 265	Fingers, cysts of (see Cysts)	
Embryo, Gasserian ganglion in	126	— fractures of, local anaesthesia in treatment	553
— growth of bone in	118	— gangrene of, due to cervical rib	557
Embryological theory of origin of frontal-sinus osteoma	569	Fistula, biliary, external, transplantation into duodenum	581
Endarteritis, proliferative, experimental production by ergot	562	— cerebrospinal, closed by facial implants	577
Endothelioma of cauda equina: report of case with removal of tumour and recovery	6	— hepato-bronchial	289
— Gasserian ganglion (see Gasserian Ganglion)		Foot, cyst of long bones of (see Cysts)	
Enterostomy, continuous feeding by, in high intestinal obstruction	467	— interarticular synovial folds in	637, 639
Epiphysis, radial, displaced, local anaesthesia in treatment	551	FORNESTER-BROWN, M.: The splinting of cases of tuberculosis of the hip	54
Erb's paralysis, sternomastoid tumour with	198	FOWLER, ANDREW: Two unusual cases of strangulated hernia	528
Ergot injections to produce proliferative endarteritis	562	Fracture of carpal scaphoid, local anaesthesia in treatment	551
Exostosis, clavicular, associated with congenital wry-neck	199	— Colles's, local anaesthesia in treatment	546, 547, 549
External biliary fistula transplanted into duodenum	581	— compression, of vertebrae	366
Extravasation of blood, subserosal, in purpura	234, 238	— of femur, local anaesthesia in treatment	551
Eye, involvement of, in frontal-sinus osteomata	571	— — spontaneous, sarcoma and	321
		— humerus, local anaesthesia in treatment	549
		— phalanges, local anaesthesia in treatment	553
		— tibia, local anaesthesia in treatment	552
		Fracture-dislocation of ankle-joint, local anaesthesia in treatment	551, 552
		Fractures of carpal and tarsal joints, intravenous anaesthesia for	646
		— in cysts of long bones of hand and foot	20, 29
		— gas gangrene complicating	38
		— infected open	294
		— — — delay in treatment	294
		— — — faulty treatment of fracture	296
		— — — mistakes in treatment	294
		— — — plaster-of-Paris in	299
		— — — plating of	298
		— — — results of treatment	295
		— — — treatment of bone fragments	297
		— — — — fracture, final	298
		— — — — primary	297
		— — — — wound, late	297
		— — — — primary	296
		— — — — Winnett Orr treatment	299
		— local anaesthesia in treatment of	546
		— — — — advantages	547
		— — — — case reports	549
		— — — — conclusions	555
		— — — — disadvantages and contra-indications	548
		— — — — dosage, etc.	553
		— — — — methods of using novocain	547
		— — — — results	554
F ACE, changes in, in congenital torticollis	199		
FACEY (John), surgeon under Henry VI	495		
Facial asymmetry, idiopathic	202		
— herpes	142, 149		
Falaise (Gilbert de), physician under Henry I	480		
Fascial implants to close cerebrospinal fistulae	577		
Feeding, continuous, by enterostomy in high intestinal obstruction	467		
Femoral artery, changes in, after periarthritic sympathectomy	216		
— hernia in a boy of 5 years	657		
Femur (see also Tuberculosis of Hip)			
— fracture of, local anaesthesia in	551		
— — spontaneous, sarcoma and	321		
— osteomyelitis of, pigmentation after use of B.I.P.P. for	329		
— sarcoma of, relation to trauma	321		

	PAGE
HAWKLEY, L. M. and BAKER, ALFRED H.: A case of primary adamantinoma of tibia - - -	415
Head (<i>see</i> Craniotomy; Intracranial; Skull)	
Heart failure, fatal, with angular deformity of spine - - -	326
— rupture of cyst into - - -	285
— surgery of (<i>see</i> Mitral Obstruction)	
Hepatic (<i>see</i> Liver)	
Hepato-bronchial fistula - - -	289
Hereditary theory of congenital torticollis - - -	188
Hermaphrodite, tubular, with teratoma of internal genitalia - - -	91
— — teratomatous enlargement of an undescended testicle - - -	335
Hernia in Africans - - -	16
— diaphragmatic, traumatic - - -	170
— femoral, in a boy of 5 years - - -	657
— strangulated, two unusual cases - - -	528
Herniation of small gut following gastrojejunostomy - - -	156
Herniotomy, gas gangrene following - - -	43
Herpes in Vth-nerve area - - -	142, 149
Heyton (Roger de), surgeon to Edward III - - -	490
Hibbs's operation and modification in tuberculosis of hip - - -	62
Hip-joint, approach to, critical review and suggested new route - - -	49
— tuberculosis of (<i>see</i> Tuberculosis of Hip)	
Hobbys (William), surgeon of Edward IV - - -	496
Hoffa-Lorenz approach to hip-joint - - -	49
Hollier (Thomas), operation on Samuel Pepys by - - -	541
Horsley's (Victor) circular skull-saw - - -	223
Hosford, Joux P.: The use of local anaesthesia in the treatment of fractures, with conclusions drawn from fifty cases - - -	546
Hour-glass stomach - - -	103
Humerus, fracture of, local anaesthesia in treatment - - -	549
Hunterian Lecture, Jan. 28, 1931, by J. Paterson Ross - - -	618
Hurst's theory of visceral tenderness - - -	377, 397
Hüter's approach to hip-joint - - -	49
Hyaline cartilage, case of osteitis fibrosa with formation of - - -	409
Hydatid cyst of liver, bleeding from portal vein during operation for - - -	585
— — rupture into biliary passages - - -	287
— — sequele of intrapleural rupture - - -	282, 284, 290
— — lung (<i>see</i> Lung)	
— — neck - - -	338
— disease, complications of - - -	275
— — clinical aspects of simple cyst - - -	277
— — development of simple cyst - - -	275
— — hepato-bronchial fistula - - -	289
— — hydatid anaphylaxis - - -	278
— — — pneumothorax - - -	292
— — rupture of cyst - - -	277

	PAGE
Hydatid disease, complications of, rupture of cyst, mechanical effects - - -	286
— — — — hepatic cyst into biliary passages - - -	287
— — — — pulmonary cyst into bronchus - - -	290
— — — — secondary echinococcosis - - -	280
— — — — — localized - - -	282
— — — — — metastatic - - -	284
— — — — — secondary cysts of peritoneum and pelvis - - -	282
— — — — — summary - - -	292
Hydronephrotic kidney, renal calculi with squamous carcinoma in - - -	590
Hypertrophic Charcot's joints, some aspects of pathology of - - -	113
I DIOPATHIC facial asymmetry - - -	202
Ileostomy, gas gangrene following - - -	43
Ileum (<i>see also</i> Intestine, Small)	
— congenital atresia of - - -	333
— cyst of - - -	521
— hemorrhage into wall of, in purpura - - -	237
Infant, unilateral polycystic kidney in - - -	162
Infants, sternomastoid tumour in - - -	190
Inguinal hernia in Africans - - -	16
Interarticular synovial folds - - -	636
Intertarsal joints, synovial folds in - - -	639
Intervertebral discs - - -	358
— — age changes in - - -	361, 370
— — anatomical observations - - -	358, 368
— — arteriosclerosis and degeneration of - - -	372
— — calcification of - - -	365, 371
— — conclusions - - -	374
— — infections of, relationship to other diseases - - -	372
— — microscopic appearances - - -	359
— — ochronosis of, in alkaptonuria - - -	370
— — osteochondritis of spine and - - -	366, 373
— — osteoporosis of vertebrae and - - -	366, 372
— — pathological changes in - - -	363, 370
— — rheumatoid and osteo-arthritis and - - -	366, 374
— — rupture of - - -	370
— — in typhoid spondylitis - - -	366, 371
— — X-ray appearances - - -	365
Intestinal bacteria in gall-bladder infections - - -	79
— obstruction complicating posterior gastrojejunostomy: case of internal strangulation of small intestine by afferent limb - - -	154
— — following subserosal extravasation of blood - - -	238
— — high - - -	467
— — administration of saline and other substances by enterostomy below obstruction - - -	467
— — cause of death in - - -	467, 475
— — rare case of cyst causing - - -	168
Intestine (<i>see also</i> Colon; Duodenum)	
— cancer of, incidence - - -	85
— non-rotation of, with congenital absence of radius - - -	166

	PAGE		PAGE
Intestine, small, congenital atresia of -	333	John, surgeon to Henry VIII -	498
— — cyst of - - - - -	521	Joints (<i>see also</i> under various joints)	
— — hæmorrhage in wall of, in purpura	234	— carpal and tarsal, lesions of intra-	
— — internal strangulation of, follow-		— venous anæsthesia for - - -	646
— — — ing gastrojejunostomy - - -	154	— Chareot's, some aspects of pathology	
— — — volvulus of, due to cyst - - -	168	— of - - - - -	113
Intra-arterial local anæsthesia - - -	641	— synovial folds in - - - - -	636
Intracranial aerocele with frontal-sinus		Jones's (Robert) approach to hip-joint -	50
— osteoma - - - - -	572	— — double hip-abduction frame for	
— — traumatic - - - - -	655	— — — tuberculous of hip - - -	61
— aneurysms - - - - -	150	Jordan of Canterbury, physician to	
— mucocele complicating frontal-sinus		— Edward III - - - - -	490
— osteoma - - - - -	572		
— tumours, post-operative mortality rate	621	KELLY, R. E. : Is it true that the	
— — radium in - - - - -	501, 618	— valves in a vein necessarily	
— — — analysis of results - - -	520	— become incompetent when the	
— — — case reports - - - - -	509, 629	— vein dilates ? - - - - -	53
— — — — cerebellar tumours - - -	514	KENDALL, E. : A rare case of intestinal	
— — — — cerebral tumours - - -	509	— obstruction - - - - -	168
— — — — pituitary tumours - - -	516	Kenleye (John de), physician in Edward	
— — — — choice of method - - - - -	503, 628	— I's reign - - - - -	486
— — — — conclusions - - - - -	520	Kidney, hæmangioma of - - - - -	308
— — — — danger of hæmorrhage - - -	631	— — — angiomata of cortex - - -	315
— — — — decompression as a prelim-		— — — — pelvic wall - - - - -	314
— — — — — inary - - - - -	503	— — — — pyramids - - - - -	315
— — — — dosage - - - - -	509, 628	— — — — ease report - - - - -	308
— — — — effect on choroid plexus	507, 632	— — — — diagnosis - - - - -	318
— — — — — normal brain tissue - - -	622	— — — — discussion - - - - -	312
— — — — experiments with rabbits -	622	— — — — factors determining hæmaturia	
— — — — immediate effects of inter-		— — — — from - - - - -	317
— — — — — stitial method - - - - -	506	— — — — review of the literature - - -	313
— — — — — surface irradiation - - -	507	— — — — summary - - - - -	319
— — — — — interstitial irradiation -	503, 628	— — — — treatment - - - - -	319
— — — — — pathological considerations -	618	— — — — hydronephrotic, renal calculi with	
— — — — — principles of irradiation -	502	— — — — squamous carcinoma in - - -	590
— — — — — results of surgical treatment -	620	— — — — polycystic, unilateral, in an infant -	162
— — — — — suggestions for future practice	633	— — — — varix of renal papilla - - -	318
— — — — — surface irradiation - - -	507, 632	King, E. S. J. : Concerning the patho-	
— — — — — use of needles or seeds - - -	503	— — — — logy of tumours of tendon-sheaths	594
— — — — — X-ray treatment - - - - -	621, 633	— — — — On some aspects of the pathology	
Intrathecal spinal tumour, unusually		— — — — of hypertrophie Charcot's joints	113
— large - - - - -	669	Koehler's approach to hip-joint - - -	50
Intrathoracic dermoid, case report -	666	Kymer (Gilbert), physician to Henry VI	496
Intravenous local anæsthesia (<i>see</i> Anæ-		Kyphosis, condition of intervertebral	
— sthesia)		— discs in - - - - -	366
Intussusception of jejunum following		— — fatal cardiac failure with - - -	326
— gastrojejunostomy - - - - -	156		
— purpura and - - - - -	238, 239	LABOUR, production of venous ob-	
Isambert, physician to Edward II -	488	— struction in - - - - -	197
Ischæmic theory of etiology of con-		Lacunar erosion of bone - - - - -	122
— genital wry-neck - - - - -	189, 198	Langenbeck's approach to hip-joint -	50
JACOBS, ARTHUR, and PRICE, L.		Larghi's approach to hip-joint - - -	50
— WOODHOUSE : Renal calculi with		Leeche (John), surgeon to Richard II -	492
— squamous carcinoma in a hydro-		Ligamentum commune, anatomy of -	427
— nephrotic kidney - - - - -	590	Ligation of veins, muscle changes fol-	
Jacques' (Frère) operation for stone -	185	— — — — lowing - - - - -	193
Jejunum (<i>see also</i> Intestine, Small)		Lip, pigmentation of, after use of	
— hæmorrhage into wall of, in purpura	234	— B.I.P.P. - - - - -	330
— strangulation of, after gastrojeunos-		Lipiodol and skiagraphy in cauda	
— — — — — tomy - - - - -	154	— — — — equina lesions - - - - -	8, 14
JENKINS, JAMES A. : Unusual complica-		Lithotomy (<i>see</i> Cutting for the Stone)	
— — — — — tion arising during operation for		Liver, hydatid cyst of, bleeding from	
— — — — — a large hydatid cyst of the liver -	585	— — — — portal vein during operation for	585

	PAGE		PAGE
Liver, hydatid cyst of, rupture into biliary passages - - -	287	Mitral obstruction, experimental - - -	259
— — — sequele of intraperitoneal rupture - - -	282, 284, 290	— — — effects on cardiac output - - -	262, 265
— non-parasitic cysts of - - -	618	— — — electrocardiographic changes in - - -	263, 265
— — — ease report - - -	618	— — — method of producing - - -	260
— — — classification - - -	651	— — — in relation to surgical treatment of mitral stenosis - - -	259
— — — clinical and pathological aspects - - -	648	— — — results of transplanting tissue in heart cavity - - -	261
— — — commentary - - -	650	— — — summary - - -	273
— — — diagnosis - - -	652	— — — X-ray appearances - - -	263, 265
— — — etiology - - -	652	— — — relief of - - -	266
— — — treatment - - -	654	— — — experiments in partial valvulotomy - - -	271
LLOYD, ENIC I.: Femoral hernia in a boy of 5 years - - -	657	— — — instrument for cutting valve - - -	268
Long bones (<i>see also</i> Fracture, etc.)		— — — technical considerations in cardiac surgery - - -	271
— — of hand and foot, cysts of (<i>see</i> Cysts)		— — — technique of partial valvulotomy - - -	269
LOVE, R. J. McNEILL: Dermoid cyst simulating gastric ulcer - - -	339	— — — widening orifice of mitral valve - - -	267
Lücke's approach to hip-joint - - -	49	Mixed tumours of bladder - - -	433, 437, 446
Lung, hydatid cysts of, rupture into bronchus - - -	290	— — salivary glands (<i>see</i> Salivary Glands)	
— — — pleural cavity - - -	292	— — — urachus - - -	433, 437, 446, 461
— — — secondary - - -	285	'Moleskin' strapping, traction by, in tuberculous of hip - - -	59
Lymphangioma, retroperitoneal - - -	523	MOLESWORTH, H. W. L.: An operation for the complete exposure of the elbow-joint - - -	303
MACALPINE, JAS. B.: Two cases of hæmangioma of the bladder - - -	205	Moleynes (James), lithotomist - - -	542
McCREA, E. D'ARCY: Tubular hermaphrodite with teratoma of the internal genitalia - - -	91	More (Marcellus de la), surgeon to Henry VIII - - -	498
Macewen's saw for cranial surgery - - -	223	MONSON, JOHN T.: Intravenous local anæsthesia - - -	641
McFADDEN, GEORGE D. F.: Retroperitoneal cysts: case of a large retroperitoneal lymph-angioma - - -	523	MONLEY, JOHN, and TWINING, E. W.: The mechanism of deep tenderness in gastric and duodenal ulcer - - -	376
Mackenzie's viscero-sensory reflex theory of visceral tenderness - - -	376	Morsted (Thomas), surgeon to Henry V - - -	493
MACREY, W. ARTHUR: Hæmangioma of the kidney - - -	308	Mouth, pigmentation of, after use of B.I.P.P. - - -	330
Marian operation for stone - - -	3	Mucocele, intracranial, complicating frontal-sinus osteoma - - -	572
Marshall (John), surgeon in Henry VI's reign - - -	496	Murphy's approach to hip-joint - - -	50
Medulloblastomata (<i>see</i> Intracranial Tumours)		Muscle changes after venous ligation - - -	193
Meningioma of Gasserian ganglion (<i>see</i> Gasserian Ganglion)		Muscles of dogs, experiments on - - -	193
Mesentery, fibroma of - - -	658	Myositis of sternomastoid muscle, wry-neck and - - -	189
Metacarpals, cysts of (<i>see</i> Cysts)		Myxochoondromatous cysts of long bones of hand and foot (<i>see</i> Cysts)	
Metastatic secondary celiinococcosis - - -	284		
Metatarsals, cysts of (<i>see</i> Cysts)			
METCALFE, R. H.: Uterus masculinus: complete tubular hermaphroditism, with teratomatous enlargement of an undescended testicle - - -	335	NAVEL (<i>see</i> Umbilicus)	
MIDDLETON, D. STEWART: The pathology of congenital torticollis - - -	188	Neck (<i>see also</i> Sternomastoid Tumour; Torticollis, Congenital)	
Migraine, differential diagnosis - - -	149	— hydatid cyst of - - -	338
MITCHELL, T. C., et al.: Studies on high intestinal obstruction: the administration of saline and other substances by enterostomy below the site of obstruction - - -	467	Needles in radium treatment of brain tumours - - -	503, 627
		Nélaton's route to hip-joint - - -	49
		NELSON, H. G. G.: A partially ossified fibroma of the stomach wall - - -	660
		Nephrectomy and nephrotomy in angioma of kidney - - -	319
		Nerves, cranial, relation to Gasserian ganglion - - -	128

	PAGE		PAGE
Nerves of main arteries, changes after peri-arterial sympathectomy -	205	Parotid gland, mixed tumours of (<i>see</i> Salivary Glands)	
Nervous theory of etiology of congenital wry-neck -	189	PATEY, D. H. : The mixed tumours of the salivary glands -	241
Neuralgia, glossopharyngeal -	149	Pelvis, secondary hydatid cysts of -	282
— trigeminal, differential diagnosis -	148	Peptic ulcer (<i>see</i> Gastric and Duodenal Ulcer)	
— — operation for -	148	Pepys (Samuel) : an historical lithotomy	541
Neuropathic arthritis, some aspects of pathology of -	113	Peri-arterial sympathectomy, anatomical result -	215
Nigarillis (David de), physician under Henry IV -	492	Peritoneum, secondary hydatid cysts of	282
Nigel, physician to William I -	480	Peroneal artery, changes in, after peri-arterial sympathectomy -	217
Nitrate of silver injections in hæmaturia	318	Peter, surgeon to Edward I -	485
Non-parasitic cysts of liver (<i>see</i> Liver)		Phalanges, cysts of (<i>see</i> Cysts)	
Non-rotation of intestine, with congenital absence of radius -	166	— fractures of, local anæsthesia in -	553
Novocain in intravenous local anæsthesia	641	— gangrene of, due to cervical rib -	557
— use in fracture treatment (<i>see</i> Fractures)		Phenol, use in miniature bone cysts -	36
		Pigmentation from bismuth absorption after use of B.I.P.P. -	329
O BSTETRICAL injuries, congenital torticollis and sternomastoid tumour and -	188, 198	Pituitary tumours (<i>see</i> Intracranial Tumours)	
Occlusion of ileum, congenital -	333	Plagiocephaly -	202
Ochronosis of intervertebral discs in alkaptonuria -	370	Plaster-of-Paris in open fractures -	299
Oligodendrogliomata (<i>see</i> Intracranial Tumours)		— tuberculosis of hip -	62
Ollier's approach to hip-joint -	50	Plating of infected fractures -	298
Orbital cavity invaded by frontal-sinus osteomata -	571	PLATT, HARRY : Cysts of the long bones of the hand and foot -	20
Orr's (Winnett) treatment of fractures	299	Pleural cavity, rupture of pulmonary cyst into -	292
OSBORNE, R. P. : The approach to the hip-joint : a critical review and a suggested new route -	49	Pneumatocele, ventricular, with frontal-sinus osteoma -	572
Ossification of intervertebral discs	366, 371	Pneumoeranium, traumatic, case report	655
Ossified (partially) fibroma of stomach wall -	660	Pneumothorax, hydatid -	292
Osteitis fibrosa, case of, with formation of hyaline cartilage -	409	POLSON, CYRIL : A case of congenital atresia of the small intestine -	333
— — miniature bone cysts and 20, 31, 35, 36		— — A case of traumatic diaphragmatic hernia -	170
Osteo-arthritis, some aspects of pathology of hypertrophic Charcot's joints -	113	Polycystic kidney, unilateral, in an infant -	162
— of spine, intervertebral discs in	366, 374	Popliteal artery, changes in, after peri-arterial sympathectomy -	217
Osteochondritis of vertebrae, intervertebral discs in -	366, 373	Portal vein, bleeding from, during operation for hydatid of liver -	585
Osteoma of frontal sinus (<i>see</i> Frontal Sinus)		Post-operative gas gangrene -	43
Osteomyelitis, acute, of spine (<i>see</i> Spine)		Posture in diagnosis of non-parasitic cysts of liver -	653
— of femur, pigmentation after use of B.I.P.P. for -	329	— relation to peptic ulcer -	378
Osteoplastic craniotomy (<i>see</i> Craniotomy, Osteoplastic)		Pott's disease of spine -	401, 402
Osteoporosis of vertebrae, intervertebral discs and -	366, 372	— — — fatal cardiac failure with -	326
Oxygen intake, etc., effect of experimental mitral obstruction on	262, 265	— — — intervertebral discs in -	367
		— fracture-dislocation at ankle-joint, local anæsthesia in treatment	551, 552
P ALATE, tumours of (<i>see also</i> Salivary Glands) -	250, 251	POWER, SIR D'ARCY : Cutting for the stone : apparatus minor and apparatus major operations -	1
Paralysis, Erb's, sternomastoid tumour with -	198	— — — Cheselden's operation -	353
		— — — Frère Jacques' operation	185
		— — — An historical lithotomy : Mr. Samuel Pepys -	541
		Preston (Simon de), surgeon to Henry III	484
		PRICE, L. WOODHOUSE, and JACOBS, ARTHUR : Renal calculi with squamous carcinoma in a hydro-nephrotic kidney -	590

	PAGE		PAGE
Ptosis, volvulus of stomach and -	105	Rare or Obscure Cases, <i>contd.</i> —	
Pugh's method of traction in tubercu- losis of hip -	59	Unilateral polycystic kidney in a child, age 1 year and 8 months -	162
Pulmonary cysts, rupture into bronchus	290	An unusually large intrathecal spinal tumour -	669
— — pleural cavity -	292	Uterus masculinus: complete tu- bular hermaphroditism, with teratomatous enlargement of an undescended testicle -	335
— — secondary -	285	Rau (J. J.) as a lithotomist -	186, 353
Purpura as an acute abdominal emer- gency -	234	Recklinghausen's disease, case with formation of hyaline cartilage -	409
— diagnosis by tourniquet test -	234	— — miniature bone cysts and 20, 31, 35, 36	
— hæmorrhage into wall of small intes- tine -	234	Recocheux (Louis), physician to Henry IV	492
— intestinal obstruction following sub- serosal extravasation of blood -	238	Rectum, cancer of, resemblance of urachal tumours to -	422, 441
— with intestinal symptoms: selecting cases for laparotomy -	239	Renal (<i>see</i> Kidney)	
— intussusception and -	238, 239	Respiration, intravenous novocain and	644
Pylorus, cancer of, duodenum involved in	84	Retroperitoneal cysts: case of a large retroperitoneal lymphangioma -	523
— in volvulus of stomach -	102, 103, 104	Reviews and Notices of Books (Titles):—	
R ABBITS. effect of radium on brain of -	622	<i>Anæsthesia and anæsthetics</i> (F. S. Rood and H. N. Webber) -	182
Radial epiphysis, displaced, local anæ- sthesia in treatment -	551	<i>Applied anatomy</i> (G. G. Davis, rev. G. P. Muller) -	342
Radio-ulnar articulation, distal, chronic subluxation of -	526	<i>L'arthrite chronique de la hanche</i> (L. Duverney) -	347
Radium therapy in duodenal cancer -	90	<i>Cancer of the lung and other intra- thoracic tumours</i> (M. Davidson)	687
— — intracranial tumours (<i>see</i> Intra- cranial Tumours)		<i>Chirurgia clinica Polonica</i> (ed. A. Jurasz and M. Rutkowski) -	179
— — mixed tumours of salivary glands	257	<i>Die Chirurgie</i> , Fascs. 26, 27 (ed. M. Kirschner and O. Nordmann) -	348
Radius, congenital absence of, with non-rotation of intestine -	166	— Fasc. 28 (ed. M. Kirschner and O. Nordmann) -	349
Radon seeds, effect on cerebral tissue of rabbits -	622	— Fasc. 29 (ed. M. Kirschner and O. Nordmann) -	538
— — in treatment of brain tumours	503, 623	<i>Die Chirurgie der Brustorgane</i> (F. Sauerbruch, ed. H. Alex- ander et al.) -	343
Rare or Obscure Cases:—		<i>Chirurgie des Kindersalters</i> (R. Draehler and J. R. Gossman) -	533
Chronic subluxation of the distal radio-ulnar articulation -	526	<i>The clinical pathology of thoracic puncture fluids</i> (S. R. Gloyne) -	350
Congenital atresia of the small intestine -	333	<i>Clinique et thérapeutique chirurgi- cales</i> (G. Pascalis) -	684
Congenital stenosis of duodenum -	331	<i>Collected papers of the Mayo Clinic</i> . Vol. xxi (ed. M. H. Mellish et al.) -	684
Cyst of the ileum -	521	<i>Congenital club-foot</i> (E. P. Brock- man) -	532
Dermoid cyst simulating gastric ulcer -	339	<i>The dawn and epic of neurology and surgery</i> (Sir C. Ballance) -	686
Femoral hernia in a boy of 5 years	657	<i>Demonstrations of physical signs in clinical surgery</i> (H. Bailey) -	180
Fibroma of the mesentery -	658	<i>Diabetic surgery</i> (L. S. McMilltrick and H. F. Root) -	347
Intracranial acrocele or traumatic pneumocephalus -	655	<i>The dramatic in surgery</i> (G. Gordon- Taylor) -	345
Non-rotation of the intestine with congenital absence of the radius	166	<i>The Edwin Smith surgical papyrus</i> (J. H. Breasted) -	341
A partially ossified fibroma of the stomach wall -	660	<i>Emergency surgery</i> (H. Bailey) -	680
Pigmentation from bismuth ab- sorption after use of B.I.P.P. -	329	<i>Handbook on tuberculosis</i> (B. S. Kanga) -	340
A rare case of intestinal obstruction	168	<i>Illustrated primer on fractures</i>	683
A rare neck cyst -	338		
Retroperitoneal cysts: report of a case of a large retroperitoneal lymphangioma -	523		
Spina bifida occulta -	172		
A thoracic dermoid -	666		
Traumatic diaphragmatic hernia -	170		
Traumatic rupture of the aorta -	664		
Two unusual cases of strangulated hernia -	528		

	PAGE		PAGE
Reviews and Notices of Books, <i>contd.</i> —		Reviews and Notices of Books, <i>contd.</i> —	
<i>Klinische Röntgendiagnostik des Verdauungskanals</i> (E. Stierlin) (H. Chaoul) - - - - -	181	<i>A student's handbook of surgical operations</i> (Sir F. Treves, rev. by C. P. G. Wakeley) - - - - -	538
<i>Di mit Block</i> - - - - -	345	<i>Surgery of the lung and pleura</i> (H. M. Davies) - - - - -	686
<i>Die Knochenbrüche und ihre Behandlung</i> (H. Matti) - - - - -	688	<i> thyroid gland</i> - - - - -	179
<i>Lehrbuch der Chirurgie</i> (ed. P. Clairmont et al.) - - - - -	537	<i>)</i> - - - - -	536
<i>Lehrbuch der Mund- und Kieferchirurgie</i> (E. Sonntag and W. Rosenthal) - - - - -	538	<i>Surgical emergencies in practice</i> (W. H. C. Romanis and P. H. Mitchiner) - - - - -	679
<i>Leonardo da Vinci, the anatomist</i> (J. P. McMurrieh) - - - - -	680	<i>Surgical pathology and morbid anatomy</i> (Bowley and Andrews) (G. Keynes) - - - - -	682
<i>Manual of surgery</i> (Rose and Carless) (C. P. G. Wakeley and J. B. Hunter) - - - - -	678	<i>A synopsis of surgery</i> (E. W. Hey Groves) - - - - -	343
<i>The modern surgical treatment of pulmonary tuberculosis</i> (B. Hudson) - - - - -	182	<i>A text-book of orthopedic surgery</i> (W. C. Campbell) - - - - -	343
<i>Orthopädie im Kindesalter</i> (H. Spitzzy and F. Lange) - - - - -	180	<i>A treatise on orthopedic surgery</i> (R. Whitman) - - - - -	683
<i> ue dans les</i> (R. Frantz) - - - - -	346	<i>The treatment of varicose veins by intravenous injections</i> (J. D. P. McLatchie) - - - - -	183
<i> Probleme in der Chirurgie</i> (C. Häbler) - - - - -	537	<i>Die Wiederbelebung: eine zusammenfassende Darstellung ihrer Theorie und Praxis</i> (O. Bruns and K. Thiel) - - - - -	349
<i>Plarr's lives of the Fellows of the Royal College of Surgeons of England</i> (rev. Sir D'Arcy Power et al.) - - - - -	674	<i>Die Wirbelbrüche und ihre Endergebnisse</i> (W. Haumann) - - - - -	347
<i>La pratique chirurgicale illustrée, Fasc. xv</i> (V. Pauchet) - - - - -	682	Authors:—	
<i>La pratique du pneumothorax thérapeutique et de la collapsothérapie chirurgicale</i> (F. Dumarest and P. Brette) - - - - -	688	Alexander, H., et al.: <i>Die Chirurgie der Brnstorgane</i> (F. Sauerbruch) - - - - -	343
<i>La rachianesthésie: sa valeur et sa place actuelle dans la pratique</i> (E. Forgue and A. Basset) - - - - -	344	Bailey, H.: <i>Demonstrations of physical signs in clinical surgery</i> - - - - -	180
<i>Radiologie clinique du tube digestif</i> (J. Gatellier et al.) - - - - -	535	— <i>Emergency surgery</i> - - - - -	686
<i>Radium in general practice</i> (A. J. Larkin) - - - - -	346	Ball, W. G., et al.: <i>St. Bartholomew's Hospital reports</i> , Vol. lxiii (ed.) - - - - -	685
<i>Radium and its surgical applications</i> (H. S. Souttar) - - - - -	181	Ballance, Sir C.: <i>The dawn and epic of neurology and surgery</i> - - - - -	686
<i>Research and medical progress and other addresses</i> (J. S. Horsley) - - - - -	344	Basset, S., and Forgue, E.: <i>La rachianesthésie: sa valeur et sa place actuelle dans la pratique</i> - - - - -	344
<i>St. Bartholomew's Hospital reports</i> , Vol. lxiii (ed. Sir T. Horder et al.) - - - - -	685	Bell, W. Blair: <i>Some aspects of the cancer problem</i> (ed.) - - - - -	534
<i>The science and practice of surgery</i> (W. H. C. Romanis and P. H. Mitchiner) - - - - -	678	Bland-Sutton, Sir J.: <i>The story of a surgeon</i> - - - - -	177
<i>Sensation and the sensory pathway</i> (J. S. B. Stopford) - - - - -	532	Block, W., and Klapp, R.: <i>Die Knochenbruchbehandlung mit Drahtzügen</i> - - - - -	345
<i>Shorter convalescence</i> (J. K. McConnel) - - - - -	686	Breasted, J. H.: <i>The Edwin Smith surgical papyrus</i> - - - - -	341
<i>A shorter surgery</i> (R. J. McN. Love) - - - - -	177	Brette, P., and Dumarest, F.: <i>La pratique du pneumothorax thérapeutique et de la collapsothérapie chirurgicale</i> - - - - -	688
<i>Some aspects of the cancer problem</i> (ed. W. Blair Bell) - - - - -	534	Brockman, E. P.: <i>Congenital clubfoot</i> - - - - -	532
<i>Stepping stones to surgery (anatomy applied to surgery)</i> (L. B. Rawling) - - - - -	682	Brown, W. L., et al.: <i>St. Bartholomew's Hospital reports</i> , Vol. lxiii (ed.) - - - - -	685
<i>Stonc, and calculous disease of the urinary organs</i> (J. Swift Joly) - - - - -	175	Bruns, O., and Thiel, K.: <i>Die Wiederbelebung: eine zusammenfassende Darstellung ihrer Theorie und Praxis</i> - - - - -	349
<i>The story of a surgeon</i> (Sir J. Bland-Sutton) - - - - -	177		

	PAGE		PAGE
Reviews and Notices of Books, <i>contd.</i> —		Reviews and Notices of Books, <i>contd.</i> —	
Campbell, W. C.: <i>A text-book of orthopedic surgery</i> - - -	343	Haumann, W.: <i>Die Wirbelbrüche und ihre Endergebnisse</i> - - -	347
Canti, R. G., et al.: <i>St. Bartholomew's Hospital Reports</i> , Vol. lxiii (ed.) - - -	685	Hewitt, R. M., et al.: <i>Collected papers of the Mayo Clinic</i> , Vol. xxi (ed.) - - -	684
Chaoul, H.: <i>Klinische Röntgen-diagnostik des Verlaunungskanales</i> (E. Stierlin) - - -	181	Holder, Sir T., et al.: <i>St. Bartholomew's Hospital Reports</i> , Vol. lxiii (ed.) - - -	685
— et al.: <i>Die Chirurgie der Brustorgane</i> (F. Sauerbruch) - - -	343	Horsley, J. S.: <i>Research and medical progress and other addresses</i> - - -	344
Clairmont, P., et al.: <i>Lehrbuch der Chirurgie</i> (ed.) - - -	537	Hudson, B.: <i>The modern surgical treatment of pulmonary tuberculosis</i> - - -	182
Davidson, M.: <i>Cancer of the lung and other intrathoracic tumours</i> - - -	687	Hunter, J. B., and Wakeley, C. P. G.: <i>Manual of surgery</i> (Rose and Carless) - - -	678
Davies, H. M.: <i>Surgery of the lung and pleura</i> - - -	686	Joly, J. Swift: <i>Stone, and calculus disease of the urinary organs</i> - - -	175
Davis, G. G.: <i>Applied anatomy</i> (rev. by G. P. Muller) - - -	342	Jurasz, A., and Rutkowski, M.: <i>Chirurgia clinica Polonica</i> (ed.) - - -	179
Denk, W., et al.: <i>Lehrbuch der Chirurgie</i> (ed.) - - -	537	Kanga, B. S.: <i>Handbook on tuberculosis</i> - - -	349
Drahter, R., and Gossman, J. R.: <i>Chirurgie des Kindersalters</i> - - -	533	Keynes, G.: <i>Surgical pathology and morbid anatomy</i> (Bowdler and Andrews) - - -	682
Dumarest, F., and Brette, P.: <i>La pratique du pneumothorax thérapeutique et de la collapsothérapie chirurgicale</i> - - -	688	Kirschner, M., and Nordmann, O.: <i>Die Chirurgie</i> , Fascs. 26, 27 (ed.) - - -	348
Duverney, L.: <i>L'arthrite chronique de la main</i> - - -	347	— — — Fasc. 28 (ed.) - - -	349
Ebert, F. M., et al.: <i>Surgical diseases of the thyroid gland</i> - - -	536	— — — Fasc. 29 (ed.) - - -	538
Evans, G., et al.: <i>St. Bartholomew's Hospital reports</i> , Vol. lxiii (ed.) - - -	685	Klapp, R., and Block, W.: <i>Die Knochenbruchbehandlung mit Drahtzügen</i> - - -	345
Felix, W., et al.: <i>Die Chirurgie des Brustorgane</i> (F. Sauerbruch) - - -	343	Lange, F., and Spitzzy, H.: <i>Orthopädie im Kindesalter</i> - - -	180
Felker, M. A., et al.: <i>Collected papers of the Mayo Clinic</i> , Vol. xxi (ed.) - - -	684	Larkin, A. J.: <i>Radium in general practice</i> - - -	346
Fitzgerald, R. R., et al.: <i>Surgical diseases of the thyroid gland</i> - - -	536	Love, R. J. McN.: <i>A shorter surgery</i> - - -	177
Forgue, E., and Basset, S.: <i>La rachianesthésie: sa valeur et sa place actuelle dans la pratique</i> - - -	344	McConnel, J. K.: <i>Shorter convalescence</i> - - -	686
Frantz, R.: <i>L'ostéo-synthèse métallique dans les fractures diaphysaires</i> - - -	346	McLachie, J. D. P.: <i>The treatment of varicose veins by intravenous injections</i> - - -	183
Gask, G. E., et al.: <i>Plarr's lives of the Fellows of the Royal College of Surgeons of England</i> (rev.) - - -	674	McMittrick, L. S., and Root, H. F.: <i>Diabetic surgery</i> - - -	347
Gatellier, J., et al.: <i>Radiologie clinique du tube digestif</i> - - -	535	McMurry, J. P.: <i>Leonardo da Vinci, the anatomist</i> - - -	680
Gloyne, S. R.: <i>The clinical pathology of thoracic puncture fluids</i> - - -	350	Matti, H.: <i>Die Knochenbrüche und ihre Behandlung</i> - - -	688
Gordon-Taylor, G.: <i>The dramatic in surgery</i> - - -	345	Mellish, M. H., et al.: <i>Collected papers of the Mayo Clinic</i> , Vol. xxi (ed.) - - -	684
Gossman, J. R., and Drahter, R.: <i>Chirurgie des Kindersalters</i> - - -	533	Mitchiner, P. H., and Romanis, W. H. C.: <i>The science and practice of surgery</i> - - -	678
Graham, E. A.: <i>Surgical diagnosis</i> (ed.) - - -	179	— — — <i>Surgical emergencies in practice</i> - - -	679
Groves, F. W. Hey: <i>A synopsis of surgery</i> - - -	183	Moulin, F., et al.: <i>Radiologie clinique du tube digestif</i> - - -	535
Haberer, H. von: <i>Lehrbuch der Chirurgie</i> (ed.) - - -	537	Muller, G. P.: <i>Applied anatomy</i> (rev.) - - -	342
Häbler, C.: <i>Physikalisch-chemische Probleme in der Chirurgie</i> - - -	537	Nordmann, O., and Kirschner, M.: <i>Die Chirurgie</i> , Fascs. 26, 27 (ed.) - - -	348
		— — — Fasc. 28 (ed.) - - -	349
		— — — Fasc. 29 (ed.) - - -	538

	PAGE		PAGE
Reviews and Notices of Books, <i>contd.</i> —		Rheumatoid arthritis, intervertebral	
Pasealis, G. : <i>Clinique et thérapeutique chirurgicales</i> - - -	684	discs and - - - - -	374
Pauehet, V. : <i>La pratique chirurgicale illustrée</i> , Fasc. xv - - -	682	Rhinorrhœa, cerebrospinal, with frontal-sinus osteoma - - -	572
Poreher, P., et al. : <i>Radiologie clinique du tube digestif</i> - - -	535	Rib, cervical, vascular complications of	557
Power, Sir D'Arcy, et al. : <i>Plarr's lives of the Fellows of the Royal College of Surgeons of England</i> (rev.) - - -	674	ROBERTSON, D. E. : A thoracic dermoid	666
Ranzi, E. : <i>Lehrbuch der Chirurgie</i> (ed.) - - - - -	537	Rodent ulcer as sequel to prolonged X-radiation - - - - -	96
Rawling, L. B. : <i>Stepping stones to surgery (anatomy applied to surgery)</i> - - - - -	682	ROGERS, LAMBERT : Osteoplastic craniotomy : a new technique for the resection and turning down of bone-flaps from the skull - -	221
Romanis, W. H. C., and Mitchiner, P. H. : <i>The science and practice of surgery</i> - - - - -	678	— — An unusually large intrathecal spinal tumour - - - - -	669
— — <i>Surgical emergencies in practice</i> - - - - -	679	Ross, J. PATERSON : The treatment of cerebral tumours with radium -	618
Rood, F. S., and Webber, H. N. : <i>Anæsthesia and anæsthetics</i> - - -	182	Rous (Adam), surgeon to Edward III -	491
Root, H. F., and McMittrick, L. S. : <i>Diabetic surgery</i> - - - - -	347	Rubber skull-cap for radium treatment of brain tumours - - -	507, 632
Rosenthal, W., and Sonntag, E. : <i>Lehrbuch der Mund- und Kieferchirurgie</i> - - - - -	538	Rupture of aorta, traumatic - - -	664
Rutkowski, M., and Jurasz, A. : <i>Chirurgia clinica Polonica</i> (ed.) -	179	— hydatid cysts (<i>see</i> Hydatid Disease)	
Sauerbruch, F. : <i>Die Chirurgie der Brustorgane</i> (ed. H. Alexander et al.) - - - - -	343	— intervertebral disc - - - - -	370
Shaw, W., et al. : <i>St. Bartholomew's Hospital reports</i> , Vol. lxiii (ed.) -	685	SALESBURY (John), surgeon to Richard II - - - - -	492
Silver, P. G., et al. : <i>Surgical diseases of the thyroid gland</i> - - -	536	Saline administered by enterostomy in high intestinal obstruction -	467
Sonntag, E., and Rosenthal, W. : <i>Lehrbuch der Mund- und Kieferchirurgie</i> - - - - -	538	Salivary glands, mixed tumours of -	241
Souttar, H. S. : <i>Radium and its surgical applications</i> - - - - -	181	— — — — clinical course of disease -	249
Spencer, W. G., et al. : <i>Plarr's lives of the Fellows of the Royal College of Surgeons of England</i> (rev.) -	674	— — — — conclusion - - - - -	258
Spitzzy, H., and Lange F. : <i>Orthopädie im Kindesalter</i> - - - -	180	— — — — correlation of clinical and histological findings -	253
Stopford, J. S. B. : <i>Sensation and the sensory pathway</i> - - - - -	532	— — — — derivation - - - - -	246
Thiel, K., and Bruns, O. : <i>Die Wiederbelebung : eine zusammenfassende Darstellung ihrer Theorie und Praxis</i> - - - - -	349	— — — — histological appearances -	242
Wakeley, C. P. G. : <i>A student's handbook of surgical operations</i> (rev.) -	538	— — — — introduction - - - - -	241
— and Hunter, J. B. : <i>Manual of surgery</i> (Rose and Carless) - - -	678	— — — — post-operative recurrence -	250
Webber, H. N., and Rood, F. S. : <i>Anæsthesia and anæsthetics</i> - - -	182	— — — — bearing on clinical course - - - - -	256
Whitman, R. : <i>A treatise on orthopedic surgery</i> - - - - -	683	— — — — — causes - - - - -	254
RHEA, LAWRENCE J., and ACKMAN, F. D. : Non-parasitic cysts of the liver : their clinical and pathological aspects - - - - -	648	— — — — — clinical types - - -	251
		— — — — — region involved - - -	250
		— — — — — relation between microscopic appearances of primary and recurrent -	257
		— — — — — terminology - - - - -	241, 249
		— — — — — treatment - - - - -	257
		Sarcoma and trauma - - - - -	321
		— of urachus (<i>see also</i> Urachus, Tumours of) - - - - -	433
		SARGENT, SIR PERCY, and CADE, STANFORD : The treatment of gliomata and pituitary tumours with radium - - - - -	501
		Sartorius muscles of dogs, experiments on - - - - -	193
		Saws for use in craniotomy (<i>see</i> Craniotomy)	
		Saxeby (Henry de), first Serjeant Surgeon - - - - -	482
		Seaphoid, carpal, local anæsthesia in treatment - - - - -	551
		SCHOFIELD, J. EWART : Carcinoma of the duodenum - - - - -	84
		Seoliosis capitis - - - - -	201

	PAGE		PAGE
Scoliosis, condition of intervertebral discs in - - - - -	636	Spongioblastomata (see Intracranial Tumours)	
— fatal cardiac failure with - - - - -	326	Spontaneous fracture in cysts of long bones of hand and foot - - - - -	20, 29
Scrotal hæmatoma after hernia operations - - - - -	18	Sputum, daughter cysts in - - - - -	289
SEARLS, H. H., et al. : Studies on high intestinal obstruction : the administration of saline and other substances by enterostomy below the site of obstruction - - - - -	467	STAMMENS, F. A. R. : A study of tumours and inflammations of the Gasserian ganglion - - - - -	125
Sensory phenomena with spinal tumour - - - - -	670	<i>Staphylococcus aureus</i> in etiology of acute osteomyelitis of spine - - - - -	400, 401
Serum treatment of gas gangrene - - - - -	47	Stenosis of duodenum, congenital - - - - -	331
Sex incidence of cysts of bones of hand and foot - - - - -	30	Stephen, surgeon of Edward II - - - - -	488
— frontal-sinus osteomata - - - - -	568	Sternomastoid muscle, blood-supply of - - - - -	197
— hernia in Africans - - - - -	17	— tumour (see also Torticollis) - - - - -	
— urachal tumours - - - - -	444	— — conclusions - - - - -	203
— volvulus of stomach - - - - -	105	— — etiology - - - - -	188, 192
SHEEN, A. W. : Osteoplastic craniotomy (introduction) - - - - -	221	— — experimental investigations - - - - -	193
Silver nitrate injections in hæmaturia - - - - -	318	— — in infancy - - - - -	190
Sinus, frontal, osteoma of (see Frontal Sinus)		— — microscopic appearances - - - - -	190
Sixth nerve, relation to Gasserian ganglion - - - - -	129, 151	— — muscle changes following venous ligation - - - - -	193
Skeletal (see also Bone)		— — production of venous obstruction in labour - - - - -	197
— traction, local anaesthesia in obtaining - - - - -	546, 549, 552	STEWART, C. P., et al. : Studies on high intestinal obstruction : the administration of saline and other substances by enterostomy below the site of obstruction - - - - -	467
— — in open fractures - - - - -	298	Stomach (see also Gastric)	
Skull (see also Craniotomy)		— hour-glass - - - - -	103
— changes in, in congenital torticollis - - - - -	199	— volvulus of (see Volvulus)	
— collection of air within - - - - -	572, 656	— wall, partially ossified fibroma of - - - - -	660
— 'plough' for osteoplastic craniotomy - - - - -	225	Stone (see Calculi ; Cutting for the Stone)	
Skull-cap for radium treatment of brain tumours - - - - -	507, 632	STORRORD, JOHN S. B., and TELFORD, E. D. : The vascular complications of cervical rib - - - - -	557
'Sliding' hernia in Africans - - - - -	17, 19	Strangulated hernia in Africans - - - - -	16
SMITH, N. Ross : The intervertebral discs - - - - -	358	— — two unusual cases - - - - -	528
Smith Petersen's approach to hip-joint - - - - -	50	Strapping, adhesive, traction by, in tuberculous of hip - - - - -	59
'Sorbo' skull-cap for radium treatment of brain tumours - - - - -	507, 632	Streptococcal infection in gall-bladder diseases - - - - -	78
Spina bifida occulta - - - - -	172	Sublingual gland, tumour of - - - - -	250, 251
Spinal tumour, intrathecal, unusually large - - - - -	669	Subluxation, chronic, of distal radio-ulnar articulation - - - - -	526
Spine (see also Intervertebral Discs)		Submaxillary glands, mixed tumours of (see Salivary Glands)	
— angular deformity of, fatal cardiac failure with - - - - -	326	Subserosal extravasation of blood in purpura - - - - -	234, 238
— excessive 'umbilication' of vertebrae - - - - -	366, 373	Sympathectomy, peri-arterial, anatomical result - - - - -	215
— osteomyelitis of, acute - - - - -	400	Synovial folds, interarticular - - - - -	636
— — case reports - - - - -	403	— membranes, normal histology of - - - - -	602
— — diagnosis from tuberculousis - - - - -	402	— 'spaces' in tendon-sheath tumours - - - - -	606
— — etiology - - - - -	401	— tuberculousis - - - - -	55, 56
— — prognosis - - - - -	402	'Synoviomias' - - - - -	594, 608
— — site of disease - - - - -	401	Syphilis, Charcot's joints and - - - - -	113
— — <i>Sta. aureus</i> and - - - - -	400, 401	— in etiology of frontal-sinus osteomata - - - - -	570
— — symptomatology - - - - -	402		
— tuberculousis of - - - - -	401, 402		
— — fatal cardiac failure with - - - - -	326		
— — intervertebral discs in - - - - -	367		
Splinting in tuberculousis of hip (see Tuberculousis of Hip)			
Spondylitis (see also Intervertebral Discs)			
— gripposa - - - - -	371		
		TALO-CALCANEAN joint, synovial folds in - - - - -	639
		TELFORD, E. D. : A case of osteitis fibrosa (with formation of hyaline cartilage) - - - - -	409

	PAGE		PAGE
TELFORD, E. D., and STOPFORD, JOHN S. B.: The vascular complications of cervical rib - - -	557	Traction in fractures, local anaesthesia in obtaining - - -	546, 549, 552
Tenderness, deep, in peptic ulcer (<i>see</i> Gastric and Duodenal Ulcer)		Transplantation of external biliary fistula into duodenum - - -	581
Tendon-sheaths, tumours of - - -	594	Trauma in etiology of frontal sinus osteomata - - -	570
— — cartilage formation and - - -	608	— — hypertrophic Chareot's disease - - -	113
— — clinical characters - - -	595	— — osteomyelitis of spine - - -	401
— — discussion - - -	604	— — sarcoma - - -	321
— — historical account - - -	594	— — tendon-sheath tumours - - -	594, 614
— — inflammatory or neoplastic origin? - - -	611	Traumatic diaphragmatic hernia - - -	170
— — macroscopic appearances - - -	596	— — pneumoeranium, case report - - -	655
— — microscopic appearances, benign varieties - - -	597	— — rupture of aorta - - -	664
— — — malignant varieties - - -	600	Trigeminal nerve area, herpes in - - -	142, 149
— — normal histology of synovial membranes - - -	602	— — lesions involving, diagnosis - - -	148
— — relationship to ganglion - - -	611	— — neuralgia, differential diagnosis - - -	148
— — summary and conclusions - - -	611	— — operation for - - -	148
— — 'synovial spaces' in tissue of - - -	606	Trigone of bladder, adenomata of - - -	437
— — terminology and classification - - -	608	Tuberculosis of hip - - -	54
'Teno-synovioma' - - -	608	— — active stage - - -	58
Tenosynovitis, tuberculous, intravenous anaesthesia for - - -	646	— — — treatment - - -	59
Teratoma of internal genitalia in tubular hermaphrodite - - -	91	— — ambulatory splint for - - -	63
Teratomatous enlargement of undescended testicle in tubular hermaphrodite - - -	335	— — case reports - - -	65
Testicle, undescended, teratomatous enlargement of, in tubular hermaphrodite - - -	335	— — clinical types - - -	56
Thoracic dermoid, case report - - -	666	— — difficulties in diagnosis - - -	55
Tibia, adamantinoma of, primary - - -	415	— — Jones's double hip-abduction frame for - - -	61
— — — clinical notes - - -	415	— — pre- and post-operative treatment - - -	77
— — — comment on case report - - -	420	— — Pugh's method of traction - - -	59
— — — pathological examination - - -	417	— — quiescent stage - - -	58
— fracture of, local anaesthesia in treatment - - -	552	— — — treatment - - -	62
Tibial artery, changes in, after periarterial sympathectomy - - -	217	— — splinting of - - -	54
Tie douloureux, differential diagnosis - - -	148	— — stage of true health - - -	58
— — operation for - - -	148	— — treatment - - -	58
Toe, fungating tumour of - - -	596	— of spine - - -	401, 402
Toes, cysts of (<i>see</i> Cysts)		— — fatal cardiac failure with - - -	326
Toilet on lithotomy - - -	3	— — intervertebral discs in - - -	367
Tongue, pigmentation of, after use of B.I.P.P. - - -	330	— — synovial - - -	55, 56
— tumour of - - -	250, 251	Tuberculous infiltration of Gasserian ganglion - - -	142
Torticollis, congenital (<i>see also</i> Sternomastoid Tumour) - - -	188	— — tenosynovitis, intravenous anaesthesia for - - -	646
— — changes in face and skull with - - -	199	Tubular hermaphrodite with teratoma of internal genitalia - - -	91
— — clavicular exostosis with - - -	199	— — teratomatous enlargement of an undescended testicle - - -	335
— — conclusions - - -	203	Tumours (<i>see under</i> various organs, etc.)	
— — morbid anatomy - - -	192	TWINING, E. W., and MORLEY, JOHN: The mechanism of deep tenderness in gastric and duodenal ulcer - - -	376
— — pathology - - -	188	Typhoid spondylitis, intervertebral discs in - - -	366, 371
— — reason for late development - - -	192		
— — skeletal changes associated with - - -	199	ULCER, gastric and duodenal (<i>see</i> Gastric and Duodenal Ulcer)	
— — and sternomastoid tumours, etiology - - -	188	— — rodent, as sequel to prolonged X-radiation - - -	96
— — — relationship between - - -	190	Ulcerating tumour of toe - - -	596
Tourniquet test in diagnosis of purpura - - -	234	Ulna, undue mobility on radius - - -	526
Toxins as a cause of death in intestinal obstruction - - -	467, 475	'Umbilication', excessive, of vertebrae - - -	366, 373
		Umbilicus, epithelial tumours near, from urachal nests - - -	459

	PAGE		PAGE
WAGNER'S osteoplastic craniotomy	222	Winnett Orr's treatment of open fractures	299
WAKELEY, CECIL P. G. : A case of fibroma of the mesentery -	658	WORSTER-DROUGHT, C., WAKELEY, CECIL P. G., ALLEN, I. M., and DICKSON, W. E. CARNEGIE : Endothelioma of the cauda equina. Report of a case with removal of tumour and recovery -	6
— — A case of unilateral polycystic kidney in a child, age one year and eight months -	162	Wounds, gas gangrene complicating, in civil surgery -	38
— — WORSTER-DROUGHT, C., ALLEN, I. M., and DICKSON, W. E. CARNEGIE : Endothelioma of the cauda equina. Report of a case with removal of tumour and recovery -	6	Wrist, fracture of, local anæsthesia in treatment -	546, 547, 549
Wareyn (Robert), physician in Henry VI's reign -	495	— ganglion and dislocations of, intra-venous local anæsthesia for -	646
WAUGH, GEORGE E. : Transplantation of an external biliary fistulous tract into the duodenum -	581	— synovial folds in -	636
Wescham (Thomas de), surgeon to Henry III -	483	Wry-neck, congenital (<i>see</i> Torticollis, Congenital)	
WHEELER, Sir W. I. DE COURCY : Pigmentation from bismuth absorption after the use of B.I.P.P. -	329	X-RAY appearances of cysts of long bones of hand and foot -	30
WHITBY, L. E. H., GORDON-TAYLOR, G. : A bacteriological study of fifty cases of cholecystectomy with special reference to anaerobic infections -	78	— — experimental mitral obstruction	263, 265
WILLAN, R. J. : Sarcoma and trauma -	321	— — gastric and duodenal ulcer -	378
William, surgeon to Henry III -	482	— — intervertebral discs -	365
WILSON, W. C. : Studies in experimental mitral obstruction in relation to the surgical treatment of mitral stenosis -	259	— — intracranial aerocele -	573, 656
		— examination of fractures, advantages of local anæsthesia in -	548
		— — in thoracic dermoid -	666
		— treatment in cerebral tumours	621, 633
		— — prolonged, rodent ulcer following -	96
		X rays and lipiodol in cauda equina lesions -	8, 14

ATLAS OF PATHOLOGICAL ANATOMY

ISSUED UNDER THE DIRECTION OF THE EDITORIAL COMMITTEE OF
The British Journal of Surgery.

FASCICULUS VI.
DISEASES OF THE JOINTS.
DISEASES OF THE THYROID GLAND.
Compiled by E. K. MARTIN, M.S., F.R.C.S.

CONTENTS

	PAGE
HÆMOPHILIC JOINTS - - - - -	I
HÆMOPHILIA - - - - -	2
The knee-joint of a child divided by sagittal section (<i>coloured</i>) <i>p.</i> 3	
ACUTE SUPPURATIVE ARTHRITIS - - - - -	4
PNEUMOCOCCAL ARTHRITIS OF KNEE - - - - -	6
A right knee-joint opened from the front (<i>coloured</i>) <i>p.</i> 7	
ANKYLOSIS OF KNEE - - - - -	8
One half of a knee-joint divided by longitudinal section (<i>coloured</i>) <i>p.</i> 9	
ACUTE SEPTIC ARTHRITIS - - - - -	10
One half of a finger divided by longitudinal section (<i>coloured</i>) <i>p.</i> 10	
TUBERCULOSIS OF THE JOINTS - - - - -	11
TUBERCULOSIS OF KNEE-JOINT - - - - -	14
One half of a right knee-joint divided by longitudinal section (<i>coloured</i>) <i>p.</i> 15	
TUBERCULOSIS OF CARPUS - - - - -	16
One half of a wrist-joint and hand divided by longitudinal section (<i>coloured</i>) <i>p.</i> 17	
TUBERCULOUS OF KNEE-JOINT - - - - -	18
One half of a left knee-joint divided by vertical section (<i>coloured</i>) <i>p.</i> 19	
TUBERCULOSIS OF KNEE-JOINT - - - - -	20
The outer half of a left knee-joint divided by longitudinal section (<i>coloured</i>) <i>p.</i> 21	
TUBERCULOSIS OF THORACIC VERTEBRÆ - - - - -	22
One half of the thoracic spine divided by sagittal section (<i>coloured</i>) <i>p.</i> 23	
Skiagram <i>p.</i> 24	
TUBERCULOUS DACTYLITIS - - - - -	25
One half of a finger divided by longitudinal section (<i>coloured</i>) <i>p.</i> 25	
TUBERCULOSIS OF ELBOW-JOINT - - - - -	26
The elbow-joint and adjacent parts of the arm and forearm of a child, divided by longitudinal section (<i>coloured</i>) <i>p.</i> 27	
TUBERCULOSIS OF SPINE - - - - -	28
One half of a cervical and upper dorsal spine, with the viscera of the neck, divided by sagittal section <i>p.</i> 29	
TUBERCULOSIS OF SPINE - - - - -	30
The lower five dorsal vertebræ, with the 1st lumbar, divided by sagittal section—surface view <i>p.</i> 31	
Section <i>p.</i> 32	

	PAGE
TUBERCULOSIS OF HIP - - - - -	33
The upper part of a child's femur divided by a section which passes through the middle of the head and neck to the base of the great trochanter <i>p.</i> 33	
TUBERCULOSIS OF SPINE (REPAIR) - - - - -	34
One half of the lower six dorsal and of the upper two lumbar vertebrae of a child, divided by vertical section <i>p.</i> 35	
ACUTE SUPPURATIVE ARTHRITIS - - - - -	36
The articular ends of a left femur and tibia (<i>coloured</i>) <i>p.</i> 37	
ACUTE SUPPURATIVE ARTHRITIS - - - - -	38
One half of a left knee divided by vertical section <i>p.</i> 39	
ACUTE SUPPURATIVE ARTHRITIS - - - - -	40
One half of a knee-joint divided by longitudinal section (<i>coloured</i>) <i>p.</i> 41	
ACUTE SUPPURATIVE ARTHRITIS - - - - -	42
The lower articular extremity of the femur <i>p.</i> 43	
FIBROUS ANKYLOSIS OF KNEE - - - - -	44
The articular surfaces of the right knee-joint torn apart (<i>coloured</i>) <i>p.</i> 45	
HEMARTHROSIS OF KNEE-JOINT - - - - -	46
The lower end of a femur, together with the patella and adjoining synovial membrane (<i>coloured</i>) <i>p.</i> 47	
GONORRHOEAL ARTHRITIS - - - - -	48
The articular surfaces of a femur and tibia from a knee-joint (<i>coloured</i>) <i>p.</i> 48	
NEUROPATHIC ARTHROPATHY - - - - -	49
CHARCOT'S DISEASE OF KNEE - - - - -	50
A left knee-joint opened by removal of its anterior wall <i>p.</i> 51	
CHARCOT'S DISEASE OF KNEE - - - - -	52
A left knee-joint opened from the front <i>p.</i> 53	
CHARCOT'S DISEASE OF KNEE - - - - -	54
One half of a right knee-joint in sagittal section <i>p.</i> 55	
ARTHRITIS DEFORMANS - - - - -	56
OSTEO-ARTHRITIS OF KNEE - - - - -	60
A left knee-joint opened from the front (<i>coloured</i>) <i>p.</i> 61	
OSTEO-ARTHRITIS OF KNEE - - - - -	62
A knee-joint laid open from the front (<i>coloured</i>) <i>p.</i> 63	
OSTEO-ARTHRITIS OF SHOULDER - - - - -	64
The head of a humerus, with a large semilunar piece of the capsule of the shoulder-joint (<i>coloured</i>) <i>p.</i> 65	
OSTEO-ARTHRITIS OF HIP - - - - -	66
The dried upper end of the right femur <i>p.</i> 67	
OSTEO-ARTHRITIS OF HIP - - - - -	68
The upper end of a right femur <i>p.</i> 68	
OSTEO-ARTHRITIS OF HIP - - - - -	69
The upper end of a left femur <i>p.</i> 69	

	PAGE
OSTEO-ARTHRITIS OF HIP - - - - -	70
The upper part of a right femur <i>p.</i> 70	
OSTEO-ARTHRITIS OF HIP - - - - -	71
One half of the upper extremity of a left femur divided by coronal section <i>p.</i> 71	
OSTEO-ARTHRITIS OF HIP - - - - -	72
The upper third of a left femur <i>p.</i> 72	
CONGENITAL DEFORMITIES OF THE THYROID GLAND - - - - -	73
THYROGLOSSAL CYST - - - - -	74
The tongue and viscera of the neck drawn from the front <i>p.</i> 75	
INFLAMMATION OF THE THYROID GLAND - - - - -	76
CYSTIC ADENOMA OF THYROID (SUPPURATION) - - - - -	77
One lateral lobe of a thyroid gland divided by coronal section (<i>coloured</i>) <i>p.</i> 77	
GOITRE - - - - -	78
MULTINODULAR COLLOID GOITRE - - - - -	83
One half of a frontal section through an enlarged thyroid gland and trachea (<i>coloured</i>) <i>p.</i> 84	
Microscopical section <i>p.</i> 85	
COLLOID ADENOMA OF THYROID - - - - -	86
An adenoma of the thyroid gland, bisected (<i>coloured</i>) <i>p.</i> 87	
COLLOID GOITRE IN ACCESSORY THYROID - - - - -	88
An oval mass of thyroid tissue three inches in vertical diameter (<i>coloured</i>) <i>p.</i> 88	
CYSTIC ADENOMA OF THYROID - - - - -	89
Part of one lateral lobe of the thyroid gland (<i>coloured</i>) <i>p.</i> 89	
CYSTIC ADENOMA OF THYROID - - - - -	90
One half of an oval swelling removed from the thyroid gland (<i>coloured</i>) <i>p.</i> 90	
COLLOID ADENOMA OF THYROID - - - - -	91
One half of an adenoma of the thyroid gland (<i>coloured</i>) <i>p.</i> 91	
CYSTIC ADENOMA OF THYROID (HÆMORRHAGE) - - - - -	92
A slice from one lateral lobe of the thyroid gland (<i>coloured</i>) <i>p.</i> 92	
CYSTIC ADENOMA OF THYROID (CALCIFICATION) - - - - -	93
Part of one lateral lobe of the thyroid gland (<i>coloured</i>) <i>p.</i> 93	
EXOPHTHALMIC GOITRE - - - - -	94
A horizontal section through the larynx and soft parts of the left side of the neck seen from below, together with a section through the aortic arch and thymus (<i>coloured</i>) <i>p.</i> 95	
Microscopical section <i>p.</i> 96	

XXI. HÆMOPHILIC JOINTS.

THE subjects of hæmophilia are always males, and the first attack of bleeding into a joint usually occurs in childhood. The knee is the chief joint affected. After a slight injury, or even with ordinary use, there is a sudden distension of the joint with blood, which stains the synovial membrane and capsule and may appear on the surface as a recognizable subcutaneous extravasation. After several attacks the joint becomes more or less permanently distended with fluid and the associated muscles waste, so that a close clinical resemblance to tuberculosis develops.

In patients who live long enough there is overgrowth of synovial fringes, degeneration of articular cartilage, and osteophytic lipping, so that the clinical picture changes to that of osteo-arthritis. Contraction of newly formed fibrous tissue in and around the ligaments of the joint often leads to flexion deformity and limitation of movement.

The X-ray appearances are normal in the early stages, and later are indistinguishable from those of osteo-arthritis.

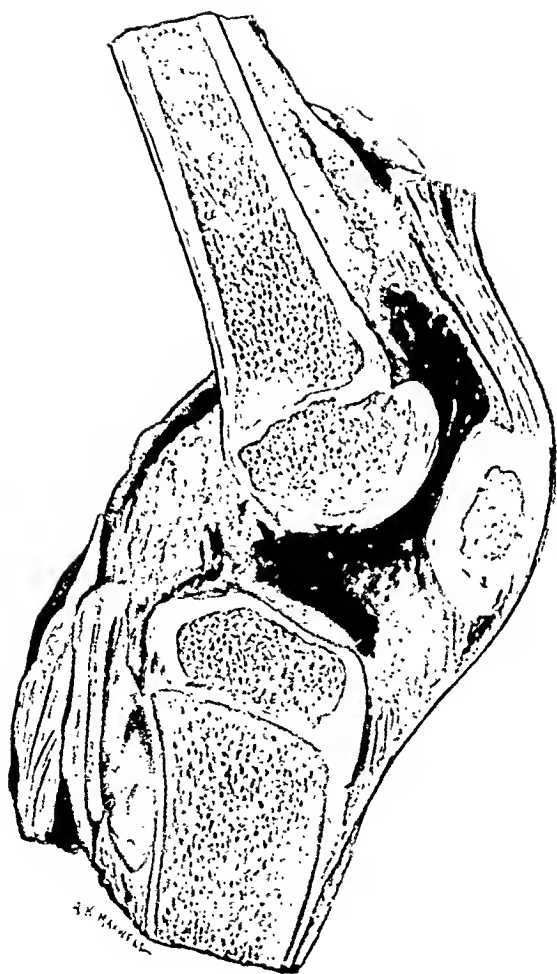
HÆMOPHILIA.

The knee-joint of a child divided by sagittal section.

The synovial cavity of the joint contains blood which has stained the synovial membrane and portions of the tibial cartilage.

Museum of St. Bartholomew's Hospital, B.225

CLINICAL HISTORY.—The patient was a boy, aged $4\frac{1}{2}$ years. His three brothers were healthy, but five maternal uncles had died from hæmophilia. The patient had been admitted to the hospital several times, on the last of which he died through hæmorrhage from a bitten tongue.



MUSEUM OF ST. BARTHOLOMEW'S HOSPITAL, B.225

XXII. ACUTE SUPPURATIVE ARTHRITIS.

THE infection may reach the joint through a penetrating wound, or along a crack communicating with a compound fracture, or by direct extension from a neighbouring focus as in acute osteomyelitis, or through the blood-stream in pyæmia. The resulting morbid changes differ only in degree. The micro-organisms are almost always of the coccal type, especially the streptococcus and the *Staphylococcus aureus*. The diplococci of pneumonia and gonorrhœa may also cause acute suppurative arthritis, and anaerobic bacteria are occasionally introduced through a penetrating wound.

At first the joint is tensely distended by a turbid effusion which contains numerous micro-organisms as well as pus cells and fibrin. In very severe infections the effusion may be blood-stained. The capsule and synovial membrane are œdematous, and the latter becomes bright red through dilatation of its vessels. Patches of fibrin adhere to its surface, and foci of necrosis mark the beginning of ulceration. The articular cartilage loses its lustre and becomes soft, particularly where the several bones of the joint exercise pressure upon one another. The softened areas of cartilage are dissolved by the proteolytic ferments in the pus, and the underlying bone is exposed. Wherever the endothelium which lines the synovial membrane and the cartilage which covers the bones are destroyed, their place is taken by granulation tissue, which erodes articular and inter-articular cartilages and the compact layer covering the ends of the bones, and then invades the cancellous spaces, the trabeculae between which become thinned by absorption. If pus is allowed to remain for long under tension in a joint, it is apt to escape through a necrotic area in the capsule either to the surface or into the soft parts around, where it tracks insidiously for considerable distances along intermuscular and fascial planes or along the sheaths of vessels.

With the release of tension the joint surfaces tend to fall together and adhere to one another, leaving pockets of pus here and there between them. Adhesion is the result of fusion of the apposed layers of granulation tissue, and ossification of this granulation tissue leads to the bony ankylosis which is so common a termination of suppurative arthritis.

For some distance from a suppurating joint the periosteum is inflamed and produces new bone. This new periosteal bone has a rough, pitted surface, and at the edge of the joint forms irregular projections from the surface (osteophytes).

The muscles which cross the inflamed joint waste more rapidly than can be accounted for by disuse only, and this wasting is probably reflex in origin. During the acute phase of the arthritis the muscles are usually in a state of contraction. This contraction, in association with relaxation of the infiltrated and distended capsule, determines the characteristic posture of joints which have become ankylosed without appropriate treatment, and, in extreme cases, may lead to pathological dislocation.

X-RAY APPEARANCES. — Destruction of articular cartilage is shown by disappearance of the normal gap between the ends of the bones. Bony changes which become visible at various stages are: rarefaction of the cancellous tissue, the formation of sequestra, deposition of inflammatory new bone beneath the periosteum, osteophytic outgrowths round the periphery of the joints, and bony ankylosis. Since the earliest X-ray signs of acute suppurative arthritis depend on destruction of cartilage, they can have

no value in clinical diagnosis during the stage in which surgical intervention is of material use in preserving function.

The above description applies to joints in which a suppurative inflammation progresses to destruction. In the more complicated articulations, such as the knee, attempts at localization of the infection by the formation of adhesions are often evident, particularly when the infection has been implanted at a single point on the articular surface, as by a penetrating wound. The adhesions are at first fibrinous, but later when cartilage has been replaced by granulation tissue, the apposed surfaces grow together so as to form a barrier between the infected and non-infected parts of the joint.

Acute suppurative arthritis may end in complete recovery with full movement if the tension is relieved early, especially when the infection is of pyæmic origin, but more commonly some degree of adhesion between the joint surfaces persists, and the most characteristic end-result is bony ankylosis. In the formation of a bony ankylosis the articular cartilage is first killed by the infection, and is then replaced by granulation tissue, which bridges the gap between the bones, and is ossified by the stream of bone cells poured out from the inflamed cancellous tissue on either side. Fibrosis or ossification in the capsule contributes to the fixation of an ankylosed joint.

Toxic Effusion.—Suppuration in the immediate neighbourhood of a joint, as in acute osteomyelitis or cellulitis, frequently determines the appearance of a synovial effusion, which may be clear or turbid, but which contains no micro-organisms, and disappears spontaneously when the causal inflammation subsides. Occasionally such an effusion may clot.

SPECIFIC TYPES OF ACUTE ARTHRITIS.

Pneumococcal Arthritis.—Occurs in children as an incident in a general septicæmia arising from the respiratory system, either direct or through suppuration in the middle ear. In adults pneumococcal arthritis is an occasional complication of pneumonia. The infection is usually limited to one of the larger joints, and is purulent in character. The severity varies within wide limits, but destructive changes with their attendant loss of function are less than in arthritis caused by the streptococcus and staphylococcus.

Gonorrhœal Arthritis.—Caused by the gonococcus, alone or in association with other micro-organisms, carried to the joint by the blood-stream from the urethra or other site of implantation. It arises usually in the third or fourth week of an attack of gonorrhœa, and affects not more than two or three joints at a time, the knee being its favourite site. The characteristic pathological lesion is an infiltration of the capsule and surrounding tissues with inflammatory exudate, which may ultimately become organized to form fibrous adhesions. In other cases a persistent or recurrent serous effusion, usually sterile, is the chief manifestation. Suppuration is rare, and is often an indication of a mixed infection of the joint. Gonorrhœal arthritis is frequently associated with non-articular forms of gonorrhœal rheumatism, such as inflammation of fascial planes, tendon-sheaths, etc.

Arthritis in Acute Specific Fevers.—Acute arthritis may develop in the course of any of the acute specific fevers, especially in typhoid and scarlet fever, measles, and small-pox. It occurs in bacillary, but not in anæmic, dysentery. Like the otitis media which so often appears under the same conditions, this variety of arthritis is caused by associated rather than by specific micro-organisms.

PNEUMOCOCCAL ARTHRITIS OF KNEE.

A right knee-joint opened from the front.

The synovial membrane of the joint is swollen and inflamed, and in many places it is creeping in a thin sheet over the articular cartilage. There are numerous synovial adhesions. Parts of the articular cartilage are eroded, and the bone, covered with red granulations, is exposed. In life such opposed areas were united by adhesions. Such articular cartilage as is left has lost its normal polish and is fibrillated. The crucial and other ligaments are hyperæmic.

Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 610/46

CLINICAL HISTORY.—The patient was a man, aged 40, who was admitted to hospital suffering from empyema after pneumonia. His knee began to swell eight days after the crisis, and he had typical starting pains at night.

At operation the pleural cavity and the knee-joint were drained. Amputation was performed eventually on account of suppuration which extended into the fascial planes of the leg and thigh. He made a good recovery.



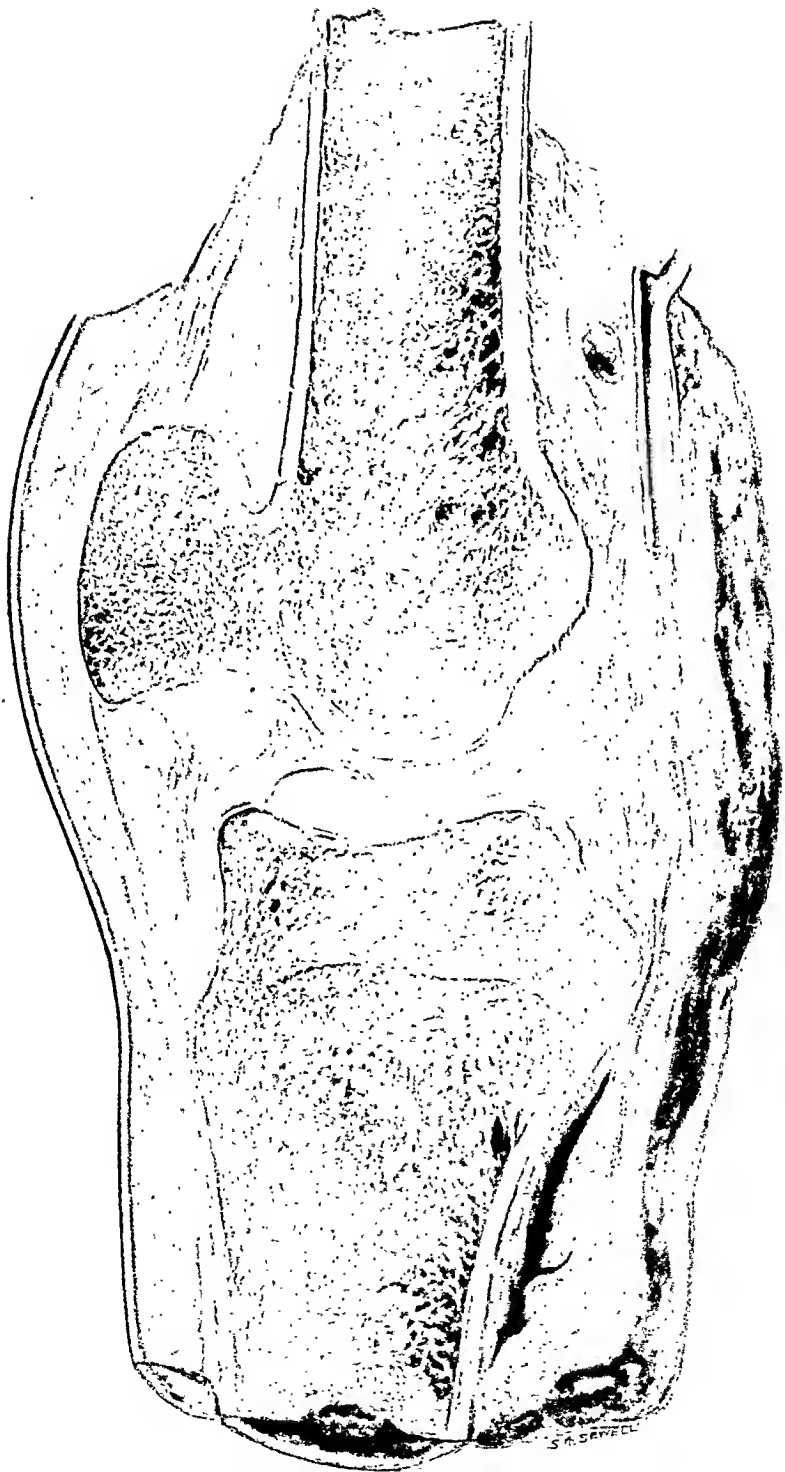
ANKYLOSIS OF KNEE.

One half of a knee-joint divided by longitudinal section.

The knee is ankylosed in a position of extension without alteration in the alinement of the bones. The ankylosis between femur and tibia is fibrous, that between femur and patella is bony. The synovial space of the joint is everywhere obliterated. The cancellous tissue of the several bones is normal in appearance, and shows no evidence of having been infected before ankylosis occurred.

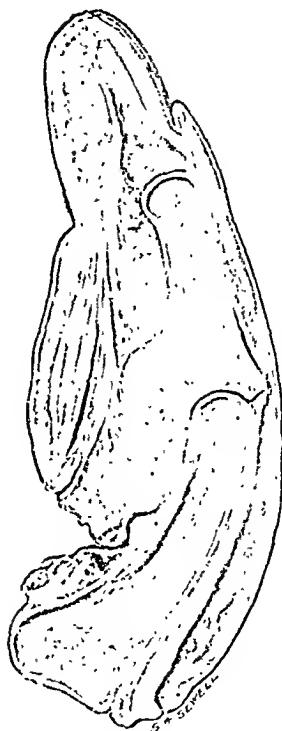
Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, C.4509

CLINICAL HISTORY.—The patient's knee became ankylosed after an operation for the removal of a semilunar cartilage.



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE, C.4509

ACUTE SEPTIC ARTHRITIS.



One half of a finger divided by longitudinal section.

The articular cartilages of the proximal interphalangeal joint have been destroyed and the two bones are in contact. The tendon of the sublimis digitorum has sloughed, and the tissues between the palmar aspect of the proximal interphalangeal joint and the tendon of the flexor profundus digitorum are swollen with œdema.

On the cutaneous surface of the finger opposite to the palmar aspect of the proximal interphalangeal joint is a small perforating wound which is not shown in the drawing.

Hunterian Museum, R.C.S., 578.1

CLINICAL HISTORY.—The finger was removed by amputation on account of its infection by a perforating wound.

XXIII. TUBERCULOSIS OF THE JOINTS.

THE disease affects both sexes at all ages, but belongs pre-eminently to childhood. A slight injury frequently precedes its appearance. The proportion of tubercle bacilli of bovine type to those of human type is much higher in tuberculosis of joints than in tuberculosis of the lungs, and this difference is connected with a different mode of entry of the micro-organisms. It is probable that in tuberculosis of joints the tubercle bacillus enters by the alimentary system, often through the drinking of tuberculous milk, and that in many cases it travels by the tonsils, through the lymphatic territories of the neck, to the blood-stream.

The first local deposit of tubercle is almost always in bone, and is usually on the diaphysial side of the epiphysial line. Occasionally the disease commences in the synovial membrane. From its original site in the bone a tuberculous focus extends to the joint by continuous invasion of the intervening tissues. This may involve perforation of the epiphysis and of the articular cartilage, or follow a more circuitous route outwards to the surface of the bone and then along the periosteum to the synovial membrane at its reflexion from bone to capsule. The passage of the disease from bone to joint is anatomically easier when the epiphysis is entirely enclosed within the capsule, as in the hip-joint.

A tuberculous focus in cancellous bone appears to the naked eye as a cavity lined by granulation tissue and containing caseous debris. The bone immediately surrounding the cavity is rarefied, and microscopic examination shows that the marrow of the cancellous spaces is replaced by tuberculous granulation tissue and that the bony trabeculae are being absorbed by osteoclasts. In this way is produced the porous appearance of the dried bone and the decrease in density of its shadow on X-ray examination. The periosteum, on the other hand, is stimulated to increased activity and lays down successive layers of new bone, as in other forms of chronic osteomyelitis.

The synovial membrane, in the early stages of infection, is studded with tubercles. Later, it becomes swollen to many times the normal thickness through infiltration by tuberculous granulation tissue, with its attendant oedema and fibrosis. The synovial surface is necrotic and the cavity of the joint may contain some tuberculous debris. An extensive fluid effusion is unusual, the main bulk of the swelling being made up of thickened synovial membrane, inflamed periosteum, and oedematous capsule.

The articular cartilage is eroded at its edges and pitted on its surface by a layer of tuberculous granulation tissue (pannus) which grows over it from the edge of the synovial membrane, and on its deep aspect it is attacked by a similar layer which detaches it from the articular end of the bone. Between the two it is softened and dissolved away. Intra-articular cartilages, tendons, and ligaments are similarly destroyed.

The capsule and ligaments of the joint become so softened by infiltration that the constant spasm of muscle which accompanies the acute stages of the disease may produce a permanent alteration in the relation of two bones to one another, as in the backward displacement of the tibia at the knee. Wasting of the muscles which normally produce movements of the joint is

a constant phenomenon in tuberculous disease. There is no associated enlargement of lymphatic glands.

In two directions a tuberculous joint may depart from the usual course of its evolution. In *tuberculous hydrops* there is an abundant serous effusion which often contains melon-seed bodies—masses of fibrin, smoothed and shaped by the movements of the joint. In *caries sicca*, which is a rare form, affecting chiefly the shoulder, the tuberculous granulation tissue which obliterates the joint cavity is small in quantity and densely fibrotic. The bones are rarefied. There is no fluid effusion.

ABSCCESS.—The necrosis of many tubercles leaves an accumulation of débris suspended in serous fluid which tends to track towards the surface of the body. The path taken by a tuberculous abscess is not necessarily the shortest route to the skin, but is rather determined by the force of gravity and conditioned by the resistance of the various anatomical structures which it encounters in its passage. Thus it may be guided by the fascial sheath of a muscle as in the psoas abscess, or along the course of vessels as in the dorsal abscess, or through such channels as the great sacrosciatic foramen.

If the joint is deeply situated, the first sign of the escape of a cold abscess from its confines is often an œdema of the overlying tissues. Later, a fluctuating swelling appears, and at any time after this the skin may become thin, red, and hot. This indicates a secondary infection of the tuberculous collection by the ordinary organisms of suppuration, and is soon followed by bursting of the abscess through the skin. The opening does not readily heal, but remains as a sinus discharging a thin, watery fluid for an indefinite period. Formation of sequestra in the rarefied area of bone, and of osteophytes around its margins, is stimulated by secondary pyogenetic infection.

SEQUESTRA.—Sequestra of various sizes may be formed in bone infected only by the tubercle bacillus, but are more common in the presence of mixed infection. As a rule they are composed of rarefied bone, but occasionally they may be densely sclerosed. A sequestrum, when loose, lies in a cavity lined by granulation tissue. The cavity may or may not communicate with the surface through a sinus.

DEFORMITY OF STRUCTURE.—The destruction of bone by tuberculous disease is commonly of such dimensions as to produce structural deformity. Examples of this are seen in the angulation of the spine in Pott's disease, and in the shortening which follows erosion of the head of the femur and of the acetabulum in tuberculosis of the hip. In addition to causing direct destruction of bone, tuberculosis leads to shortening by interference with the growth from epiphysal cartilage, and frequently induces a postural deformity through coincident contracture of muscles.

DISABILITY OF FUNCTION.—In the active stage of tuberculosis loss of mobility is due to pain and muscular spasm. Later it is caused by wasting of muscles, by destruction of articular cartilage, by alterations in the shape and length of the bones, and by fibrous adhesions within and around the joint.

ANKYLOSIS.—Fibrous ankylosis is the usual result of uncomplicated tuberculosis. When there has been mixed infection, with sinuses, bony ankylosis is common. In either case, foci of living tubercle bacilli are apt to become encapsuled in the joint, and these may again become active, after many years of quiescence, in consequence of accidental or operative injury.

TUBERCULOSIS OF SPECIAL JOINTS.

Spine.—The primary focus is usually in the front of the body of the vertebra immediately beneath the epiphysis or the periosteum, and as a rule more than one vertebra is involved. The disease destroys the vertebral bodies affected, together with the intervening discs, and, with their dissolution, the parts of the column above and below fall together to produce an angular deformity.

Abscesses are common, and, in general, tend to track downwards under the influence of gravity. In the neck a retropharyngeal abscess passes laterally behind the carotid sheath and presents in the posterior triangle. In the thorax a mediastinal abscess may be recognized by the X rays. From the lower dorsal and the lumbar spine a psoas abscess passes down in the sheath of the muscle and may present above or below Poupart's ligament; or it may cross the brim of the pelvis and emerge through the great sacro-sciatic foramen into the buttock. From any part of the spine a dorsal abscess may accompany the dorsal vessels through the erector spinæ. A lumbar abscess may form behind the kidney.

The spinal cord may be compressed either by an abscess or by tuberculous granulation tissue which has invaded the extradural portion of the spinal canal.

Healing of spinal caries is by bony ankylosis.

Hip.—The primary focus is usually in the neck of the femur immediately distal to the epiphysal cartilage. Occasionally it is related to the Y-shaped epiphysal cartilage of the acetabulum. Destruction of bone is greatest at the points of greatest pressure—i.e., the upper surface of the femoral head and the upper and back part of the acetabulum. In the latter situation erosion is compensated by the production of new periosteal bone, so that dislocation from this cause is uncommon.

Abscesses present forwards in the outer part of Scarpa's triangle, backwards beneath the gluteals, and inwards through the floor of the acetabulum into the pelvis. Sequestra are common. There is often considerable restoration of function; if ankylosis occurs, the ultimate deformity tends to be one of flexion, adduction, and internal rotation, with compensatory lordosis and scoliosis.

Knee.—The primary focus may be in bone or in synovial membrane, and in either case thickening of the latter forms a prominent feature of the disease. Sinus formation is common, but extensive abscesses are rare, and are limited to debilitated patients in whom the course of the disease is very rapid. In such a case an abscess may pass up beneath the quadriceps and fill the thigh.

Two uncommon types of tuberculous arthritis are met with in the knee, one characterized mainly by effusion (*tuberculous hydrops*), the other by a nodular thickening of the synovial membrane (*Koenig's type*) in addition to effusion.

Healing of a tuberculous knee may be accompanied by complete or partial restoration of function; but fibrous ankylosis is common. Deformity, if present, comprises flexion, external rotation, and backward displacement of the tibia on the femur.

TUBERCULOSIS OF KNEE-JOINT.

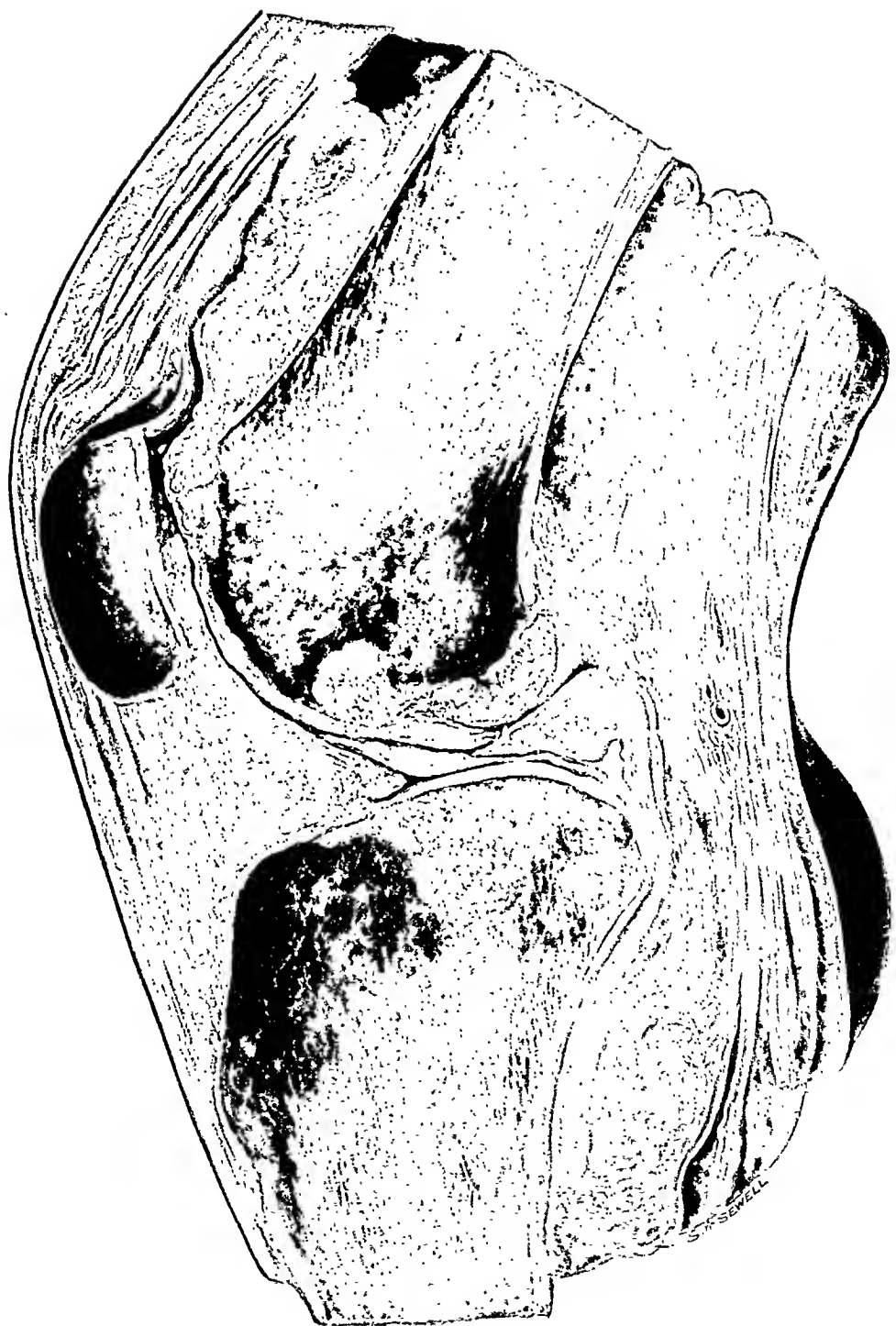
One half of a right knee-joint divided by longitudinal section.

The joint is swollen and the tibia is displaced slightly backwards upon the femur. Immediately beneath the articular cartilage covering the opposed parts of the femur and tibia are foci of caseous tuberculosis. The articular cartilages of the several bones are destroyed in places, chiefly over the anterior and posterior extremities of the femoral condyle. Undermining by tuberculous granulation tissue of the articular cartilage of the patella, and erosion of its surface, have just commenced.

The synovial membrane is thickened by tuberculous granulation tissue throughout its whole extent, and the cavity of the joint is distended by the débris of caseating tubercle. On its posterior aspect an extension of the joint lined by tuberculous granulation tissue passes down for three inches between the tibia and the popliteal vessels.

Hunterian Museum, R.C.S., 4619.1

CLINICAL HISTORY.—The patient was a man, aged 67, who suffered from chronic pulmonary tuberculosis for two years before the date of amputation. His knee had been enlarging and becoming increasingly painful, and had produced profound constitutional disturbance.



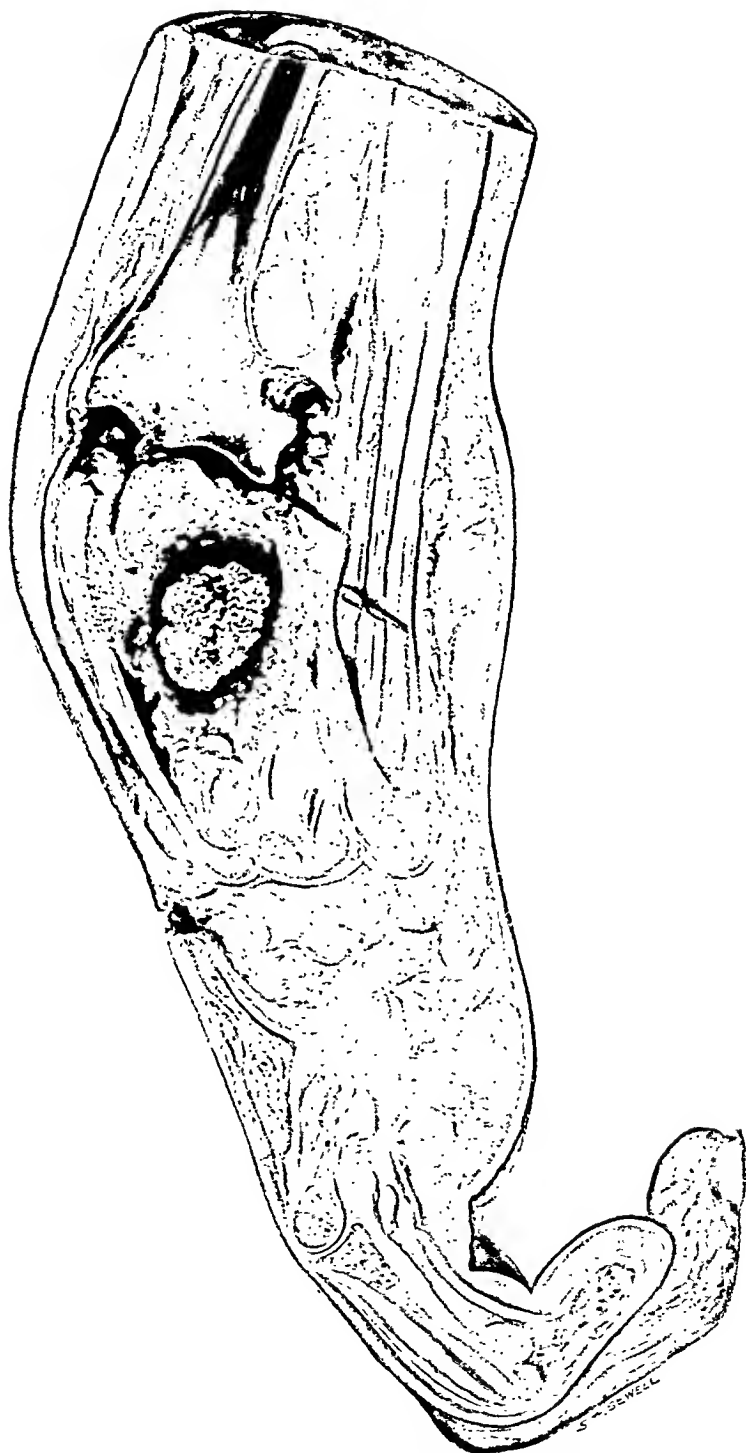
TUBERCULOSIS OF CARPUS.

One half of a wrist-joint and hand divided by longitudinal section.

The carpus is the seat of advanced tuberculous osteitis, and shows a cancellous sequestrum lying in an abscess cavity. The articular cartilage of the wrist-joint has been destroyed and is replaced by tuberculous granulation tissue, an extension of which spreads beneath the flexor tendons of the forearm. The skin on the volar surface of the wrist is perforated by sinuses which communicate with the abscesses seen on the ext surface. Another sinus, which is seen in section, opens on the dorsum of the hand. The wrist, fingers, and thumb are held in a position of flexion.

Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 605/20

CLINICAL HISTORY.—The specimen was removed by amputation from a man aged 25.



TUBERCULOSIS OF KNEE-JOINT.

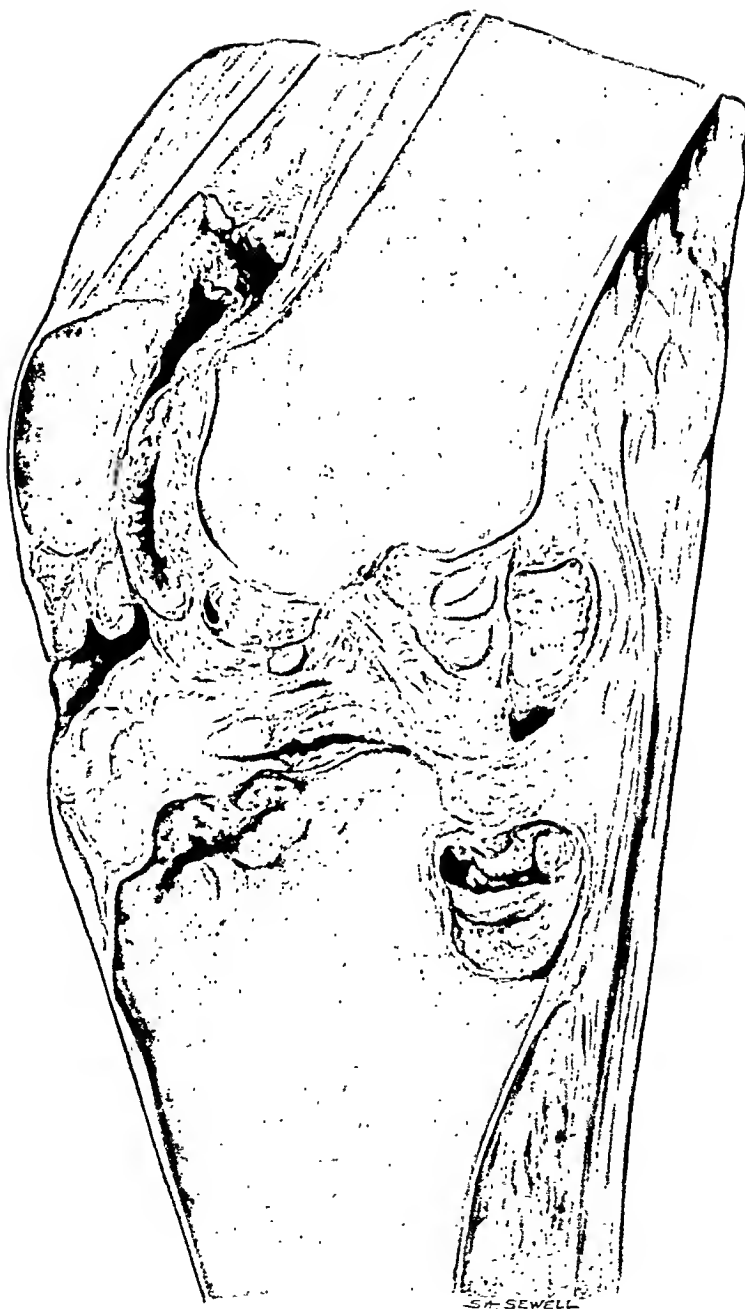
One half of a left knee-joint divided by vertical section.

In the anterior part of the head of the tibia just below the articular surface is an irregular cavity filled with caseating granulation tissue. This probably represents the primary lesion from which the synovial membrane of the joint was infected. There is a second larger cavity similar in type in the posterior part of the head of the bone. The popliteal space contains caseating material. The articular cartilage of all three bones has been replaced by tuberculous granulation tissue. In the recent state the cavity of the joint contained creamy pus.

Hunterian Museum, R.C.S., 892.1

MICROSCOPIC STRUCTURE.—Caseating giant-cell systems.

CLINICAL HISTORY.—The patient was a soldier, aged 30, whose left testis was removed for tuberculosis in India. He returned to England and developed caries of the spine with bilateral psoas abscess, and tuberculosis of the left knee-joint. The left lower limb was amputated eighteen months after the appearance of the disease. He died four years after the commencement of his illness.



HUNTERIAN MUSEUM, R.C.S., 892.1

TUBERCULOSIS OF KNEE-JOINT.

The outer half of a left knee-joint divided by longitudinal section.

In the lower end of the femur there is a cavity lined with granulation tissue and containing a sequestrum. Most of the articular cartilage has been destroyed and replaced by granulation tissue. The cavity of the joint has not been obliterated. The surrounding muscles are extensively replaced by fat.

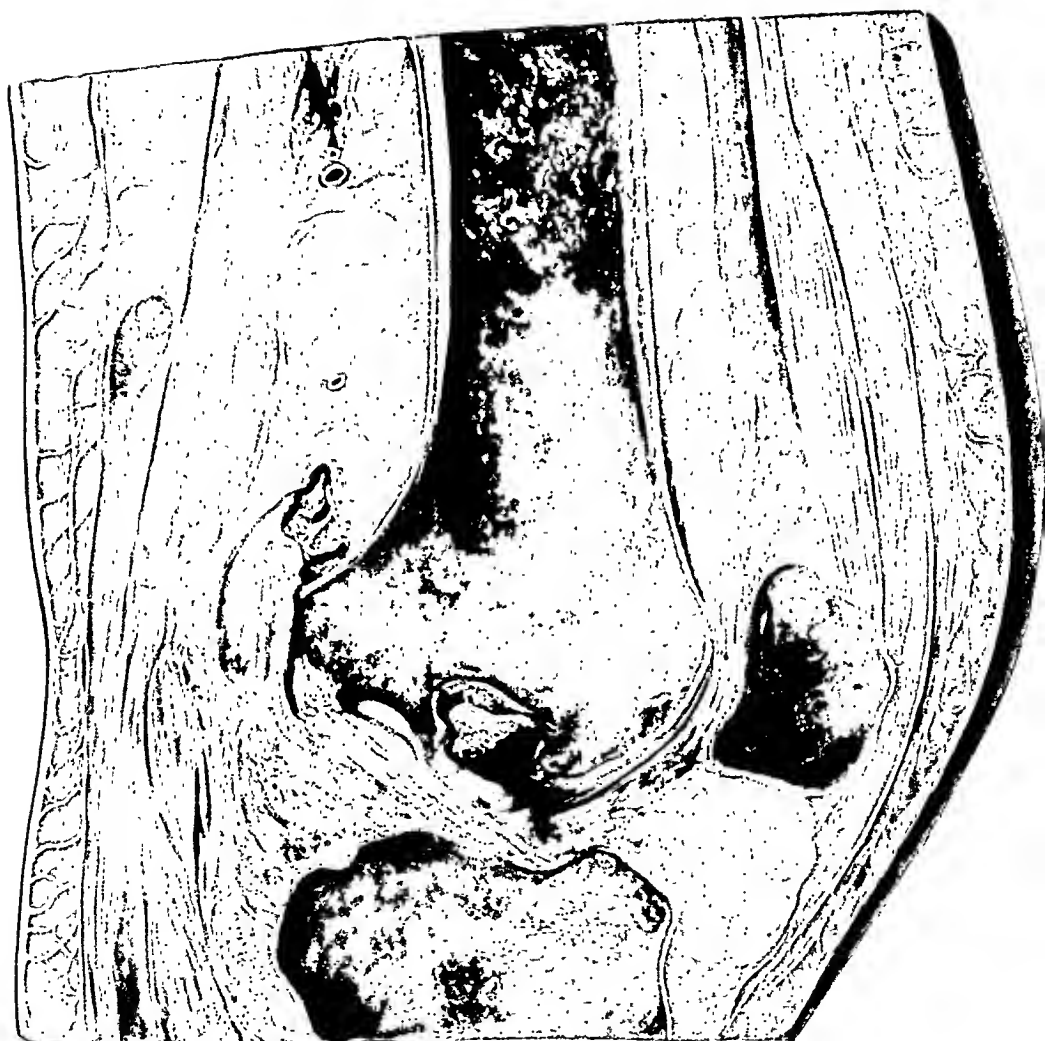
At the back of the specimen is a sinus leading down to the joint. Near the head of the fibula is a fragment of a needle.

Hunterian Museum, R.C.S., 900.2

CLINICAL HISTORY.—The patient was a middle-aged woman who, in 1903, knelt on a cushion and ran a needle into the soft parts to the inner side and below her left patella. In 1908 the needle was broken by gymnastic exercises, but there were no serious ill effects. In 1910 the knee became painful. In 1911 the joint increased in size and the diagnosis of tuberculosis was made. The broken needle was demonstrated by X rays and an unsuccessful attempt was made to remove it. The limb was placed on a splint, and was moved from time to time with the object of breaking down adhesions.

In 1914 a sinus opened on the inner side of the patella at the site of the operation scar. Other sinuses appeared later, and tubercle bacilli were demonstrated in the pus. In 1915 the leg was amputated.

The needle had been used by a phthisical person who had been in the habit of placing the eyes of the needles in her mouth.



S.F. SEWELL

HUNTERIAN MUSEUM, R.C.S., 900.2

TUBERCULOSIS OF THORACIC VERTEBRÆ.

One half of the thoracic spine divided by sagittal section.

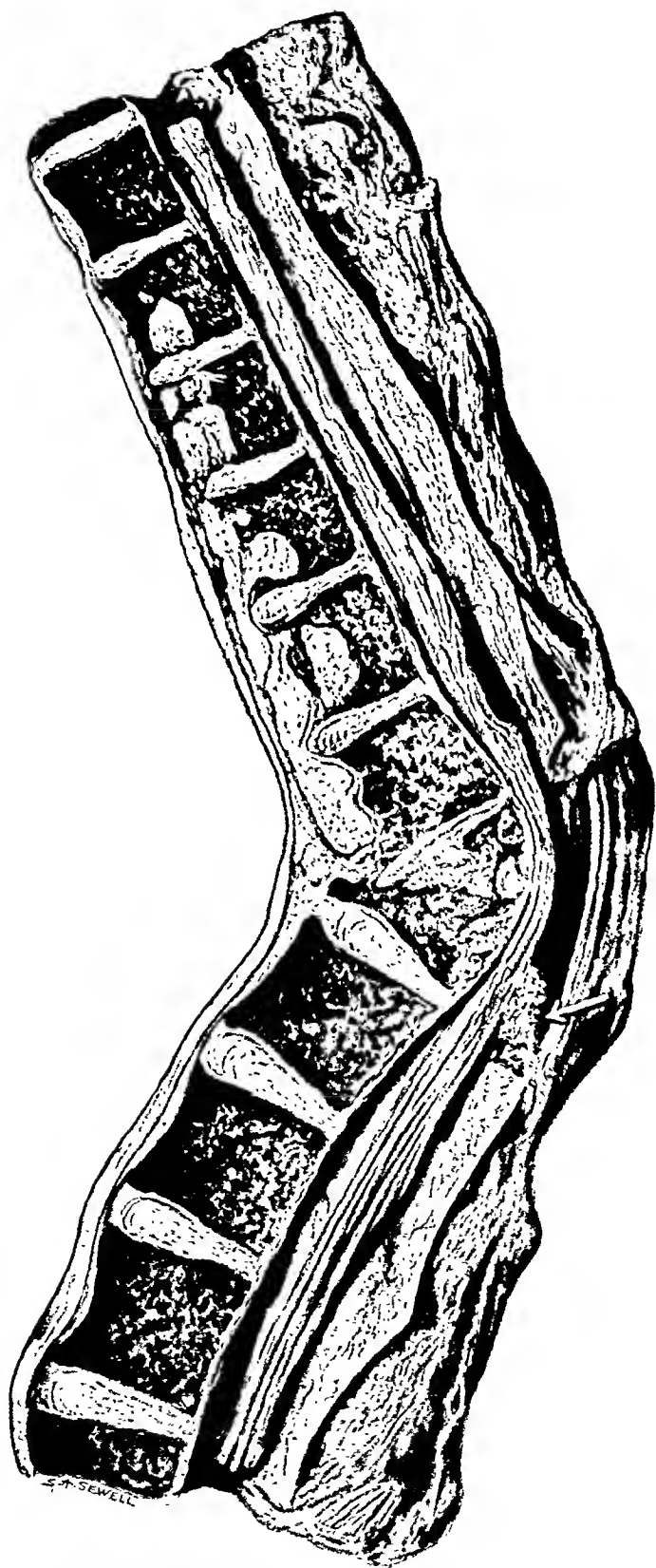
The body of one of the lower thoracic vertebræ is almost completely destroyed by tuberculous osteitis, so that an acute angular deformity has been produced. The laminæ and spinous processes at the site of the curve have been removed by operation. The cord was compressed at the point indicated by the arrow. A tuberculous abscess spreads up the anterior surface of the bodies of the vertebræ under the anterior common ligament. There are secondary tuberculous foci in the bodies of the vertebræ above the primary lesion. On the reverse side of the preparation there was a large abscess at the side of the spine.

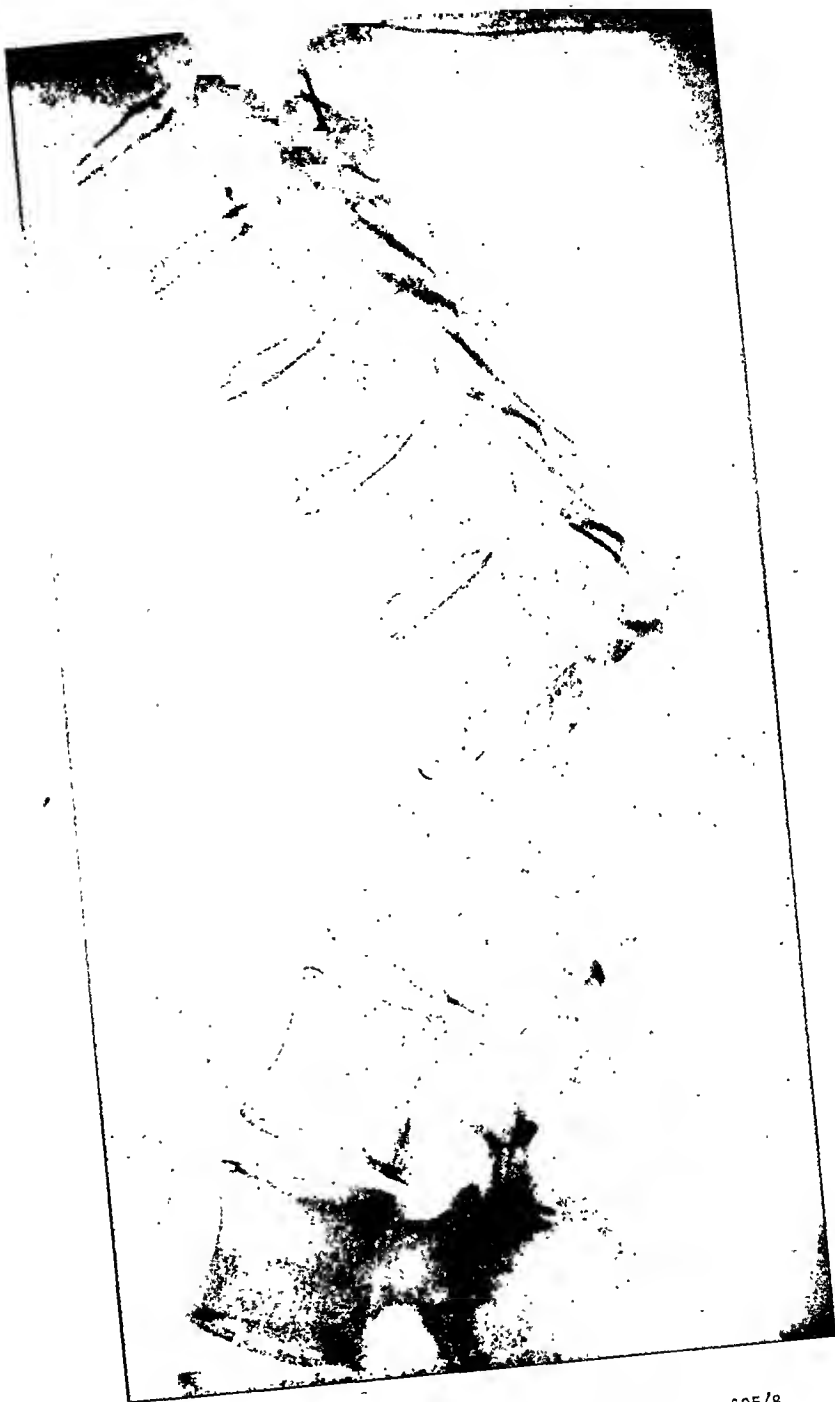
Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 605/8

CLINICAL HISTORY.—The patient was a man, aged 23, who was admitted to hospital with spinal caries and paraplegia. Laminectomy was followed by considerable benefit, but he died from generalized tuberculosis four months later.

X-RAY.—The whole of the body of the twelfth dorsal vertebra and the greater part of the body of the first lumbar vertebra are destroyed. From this point upwards there is a progressively diminishing absorption of the bodies of the next five dorsal vertebræ.

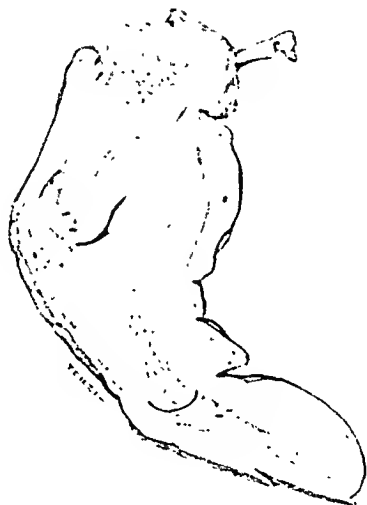
The dorsal column has bent forwards so as to form an angle of about 100° with the lumbar column. At the site of the greatest destruction the spinous processes of the two most damaged vertebræ project backwards.





MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE, 605/8

TUBERCULOUS DACTYLITIS.



One half of a finger divided by longitudinal section.

The finger is considerably swollen, and the head of the proximal phalanx is destroyed by caries. There is a white opaque area of caseous tuberculosis in its cancellous tissue, and there are similar foci in the soft tissues around the joint both on the flexor and the extensor surfaces. The interphalangeal joint has escaped infection.

Hunterian Museum, R.C.S. 877.2

CLINICAL HISTORY.—The patient was a man who suffered from pulmonary tuberculosis.

TUBERCULOSIS OF ELBOW-JOINT.

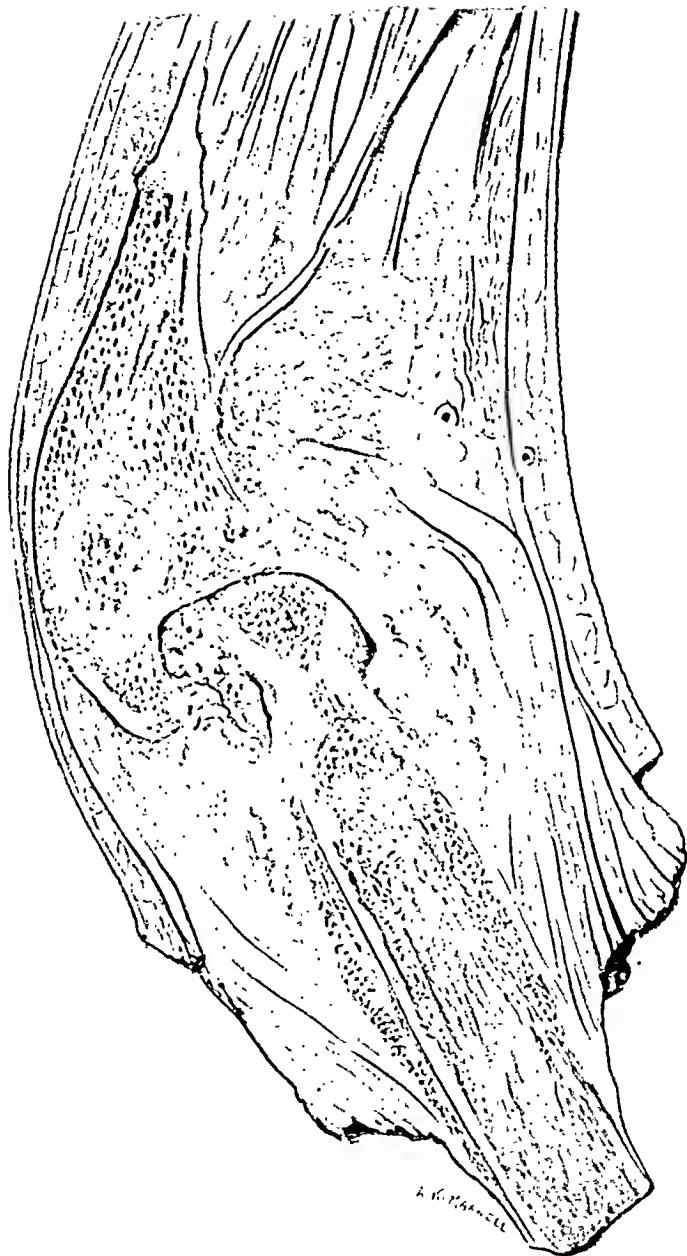
The elbow-joint and adjacent parts of the arm and forearm of a child, divided by longitudinal section.

The elbow is swollen and there is a sinus in the skin over the olecranon. The ulna is displaced backwards from its normal relation to the humerus. The upper end of the ulna is thickened by the deposit of new periosteal bone on its posterior surface and contains a cavity lined by granulation tissue and filled with caseous material.

The lower part of the shaft of the humerus is similarly thickened by new periosteal bone. The articular cartilage of the humero-ulnar portion of the elbow-joint has disappeared. The synovial membrane is thickened and the cavity of the joint is occupied by caseous material.

Museum of University College Hospital, 20.B.1

No clinical history.



TUBERCULOSIS OF SPINE.

One half of a cervical and upper dorsal spine, with the viscera of the neck, divided by sagittal section.

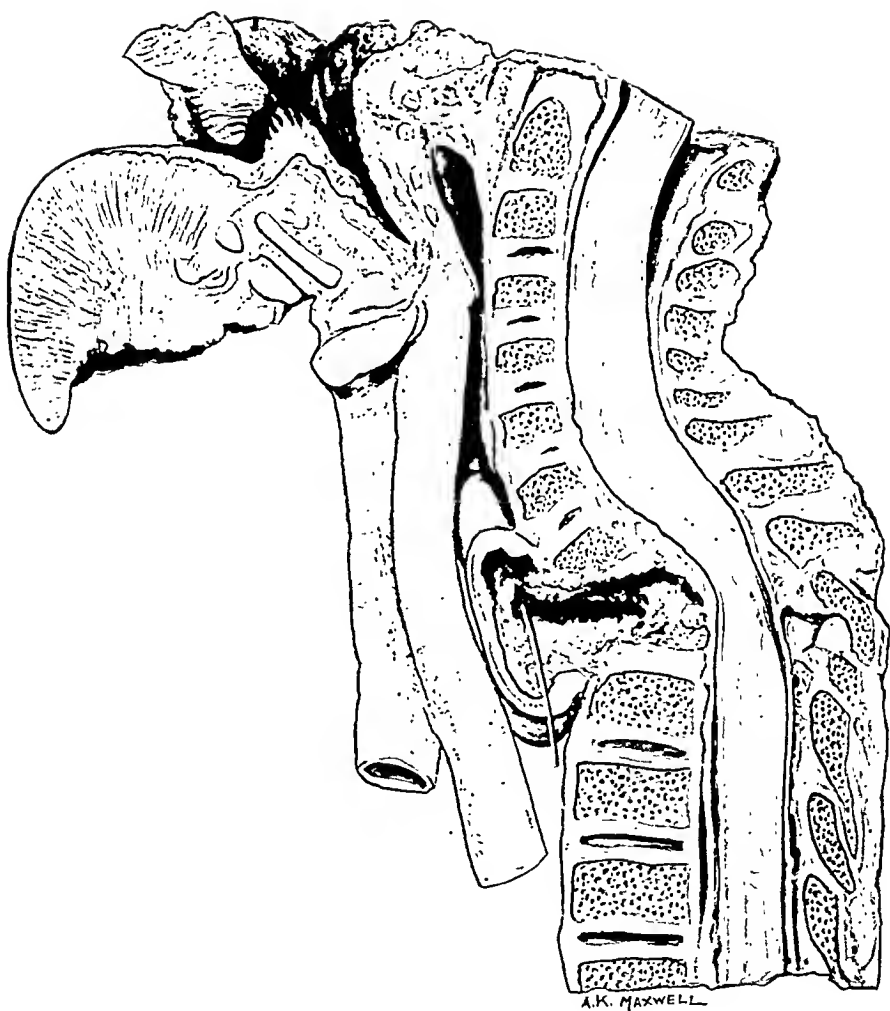
The spine is bent at the junction of the cervical and dorsal regions so as to be convex backwards, the second dorsal spinous process being the most prominent. The upper five dorsal vertebral bodies have been destroyed in whole or in part. The cavity resulting from their destruction is lined by tuberculous granulation tissue and is continued anteriorly into a thick-walled abscess which pushes forward the œsophagus and trachea. From its right side extensions pass up along the front of the transverse processes of the cervical vertebræ behind the carotid sheath, and downwards to encroach on the upper part of the right pleural cavity.

The spinal canal is narrowed and the cord compressed by granulation tissue which surrounds the thickened theca. The root of the fourth dorsal spinous process has been destroyed, and in the interval between the second and third processes a small abscess extends into the muscles of the back.

Museum of University College Hospital, 16.B.2

CLINICAL HISTORY.—The patient was an emaciated boy, aged 4 years, whose head fell forwards so that his chin rested on the sternum and respiration was embarrassed. He died six days after admission to hospital.

AUTOPSY.—Bronchopneumonia. Mediastinal glands caseous; left pleural cavity obliterated.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 16.B 2

TUBERCULOSIS OF SPINE.

The lower five dorsal vertebræ, with the 1st lumbar, divided by sagittal section.

The disc between the 11th and 12th dorsal vertebræ has been destroyed with the greater part of the adjacent epiphysial plates. The apposed surfaces of the bodies and the anterior surface of the upper one are ulcerated, and the superficial layers of cancellous bone are filled with granulation tissue.

An oval abscess occupies the left aspect of the affected vertebral bodies and communicates with the space between them. The spinal canal is normal.

Museum of University College Hospital, 14.B.2

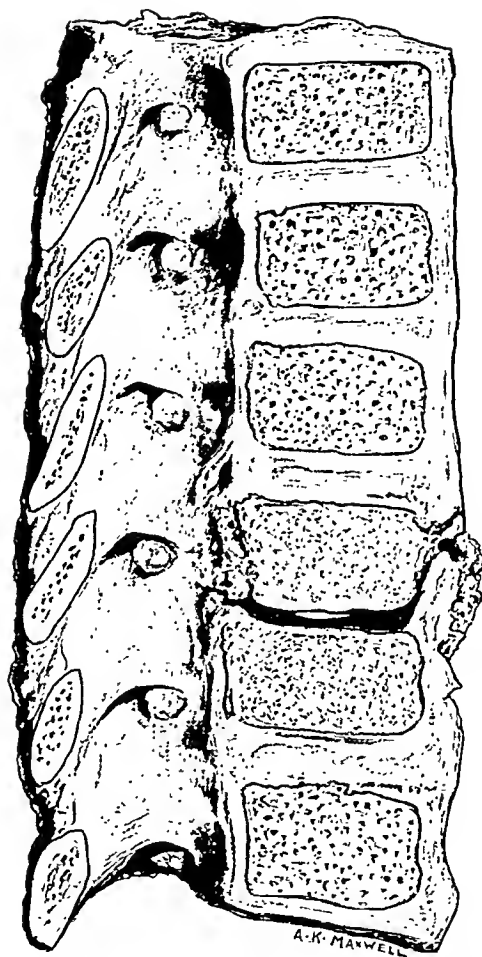
CLINICAL HISTORY.—The patient was a boy, aged 3 years, who had suffered from pain in the back for three weeks. On admission to hospital there was angulation of the dorsal spine. One week after admission there was a rise of temperature with vomiting, squint, and convulsions. He died in ten days.

AUTOPSY.—Tuberculous meningitis at base of brain. Miliary tuberculosis of lungs. Caseous mediastinal and mesenteric glands.



SURFACE.

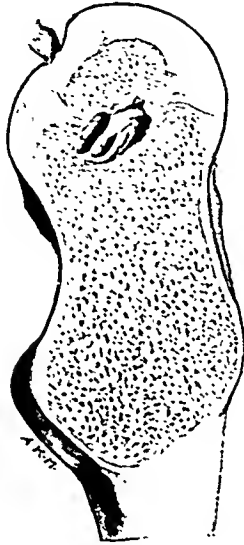
MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 14.B.2



SECTION.

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 14.B.2

TUBERCULOSIS OF HIP.



The upper part of a child's femur divided by a section which passes through the middle of the head and neck to the base of the great trochanter.

The centre of the epiphysial cartilage of the head and the adjacent cancellous bone of the neck have been destroyed. At this point there is a cavity lined by granulation tissue and containing a small sequestrum.

The bone forming the floor of the non-articular part of the acetabulum is soft, and on its pelvic aspect is thickened by a deposit of new periosteal bone.

Museum of University College Hospital. 23.B.1

CLINICAL HISTORY.—The patient was a child who died of tuberculous meningitis.

TUBERCULOSIS OF SPINE.

(REPAIR.)

One half of the lower six dorsal and of the upper two lumbar vertebræ of a child, divided by vertical section.

The column presents a sharp angular curvature as a result of destruction of the greater part of the bodies of the lower three dorsal and of the 1st lumbar vertebræ.

The carious process has come to an end, and the remnants of the bodies of the affected vertebræ have become firmly ankylosed by bone.

The vertebral canal is not diminished in calibre.

Hunterian Museum, R.C.S. 890.1

No clinical history.



HUNTERIAN MUSEUM, R.C.S. 890.1

ACUTE SUPPURATIVE ARTHRITIS.

The articular ends of a left femur and tibia.

The articular cartilage has been replaced by granulation tissue over the opposed surfaces of the two bones. The synovial membrane is red and œdematous. The anterior parts of both semilunar cartilages have been destroyed.

Hunterian Museum, R.C.S. 681.5. Army Medical Collection.

CLINICAL HISTORY.—The patient was a man whose left knee was infected through a penetrating gunshot wound. The joint was drained for a month, at the end of which the limb was amputated on account of the spread of suppuration along the hamstrings.



HUNTERIAN MUSEUM, R.C.S. 681.5. ARMY MEDICAL COLLECTION

ACUTE SUPPURATIVE ARTHRITIS.

One half of a left knee divided by vertical section.

The joint is swollen and the tibia is displaced backwards upon the femur. The articular cartilage of the several bones has been destroyed and the surrounding soft parts are infiltrated by inflammatory exudate. At the back of the joint pus has escaped through the posterior ligament and has formed abscesses behind both tibia and femur.

On the outer side of the joint is a vertical granulating wound, at the bottom of which the external condyle of the femur is exposed. It is not recorded whether this wound was made in the course of treatment, or whether it is a result of the injury which led to infection of the joint.

Hunterian Museum, R.C.S. 660.1

CLINICAL HISTORY.—The patient was a man, aged 60, who when suffering from suppurative arthritis refused to have the pus evacuated. The joint rapidly became disorganized and the leg displaced backwards. The limb was amputated six weeks after the commencement of the arthritis.



HUNTERIAN MUSEUM, R.C.S. 660.1

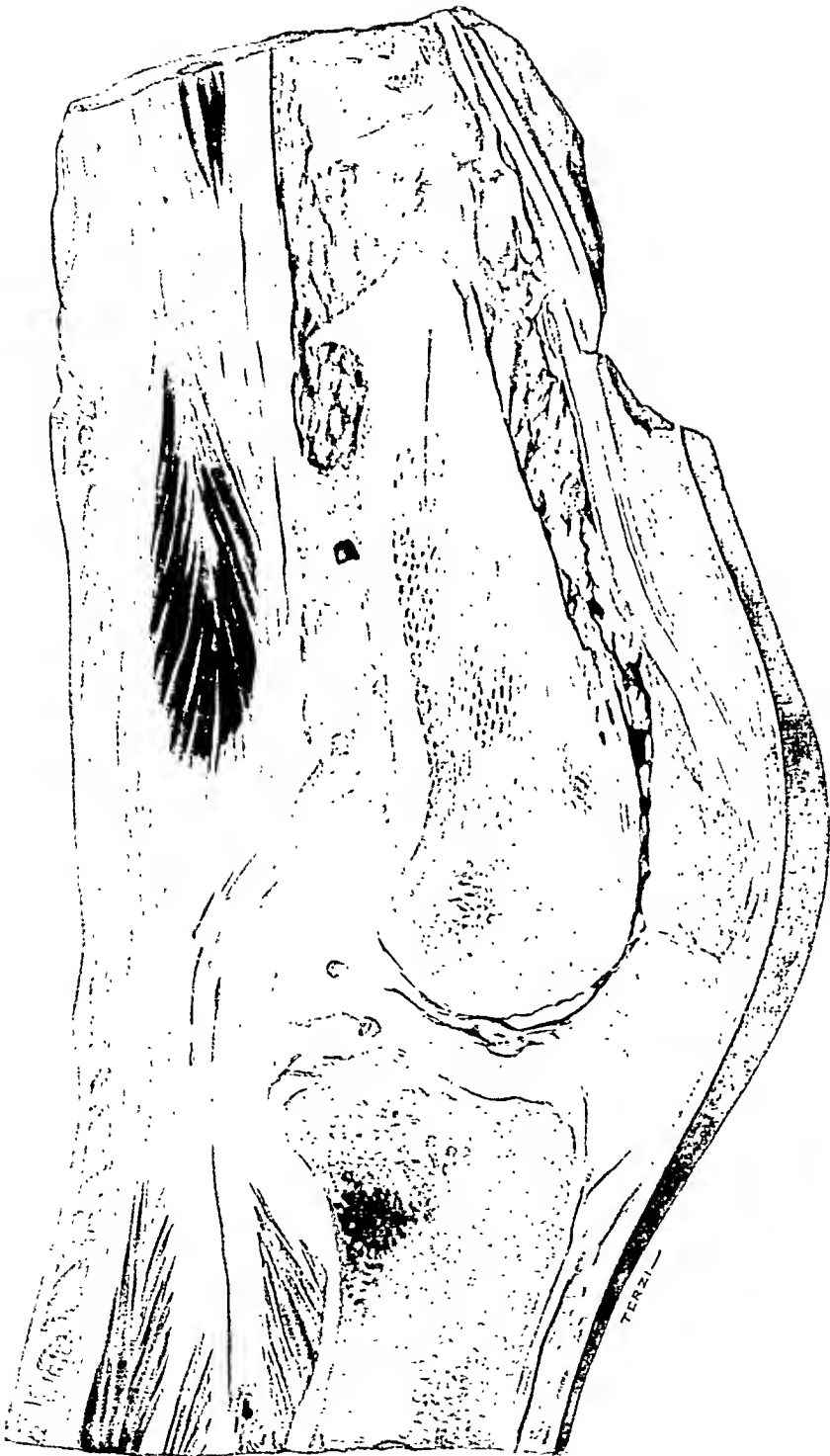
ACUTE SUPPURATIVE ARTHRITIS.

One half of a knee-joint divided by longitudinal section.

The articular cartilage of the several bones which enter into the knee-joint has been destroyed in some parts, whilst in others it is undermined by granulation tissue. The synovial membrane is coated with pus, which in the fresh state distended the joint. The pus has escaped from the joint cavity at the upper limit of the suprapatellar pouch and has extended for several inches along the femur beneath the crureus.

Hunterian Museum, R.C.S. 660.2

CLINICAL HISTORY.—The patient was a man, aged 60, who suffered from a chronic, painful, and gradually increasing swelling of the knee-joint. It was thought to be secondary to pyorrhœa, and the affected teeth were removed. A vaccine was made from a micrococcus which was thought to be the cause of the pyorrhœa, and with this the patient was treated. Aspiration of the knee-joint yielded a thin pus containing cocci resembling those from the gums, but all attempts to cultivate them failed. As the condition of the limb became worse, amputation was performed and the patient made a good recovery, although the flaps united slowly.



HUNTERIAN MUSEUM, R.C.S. 660.2

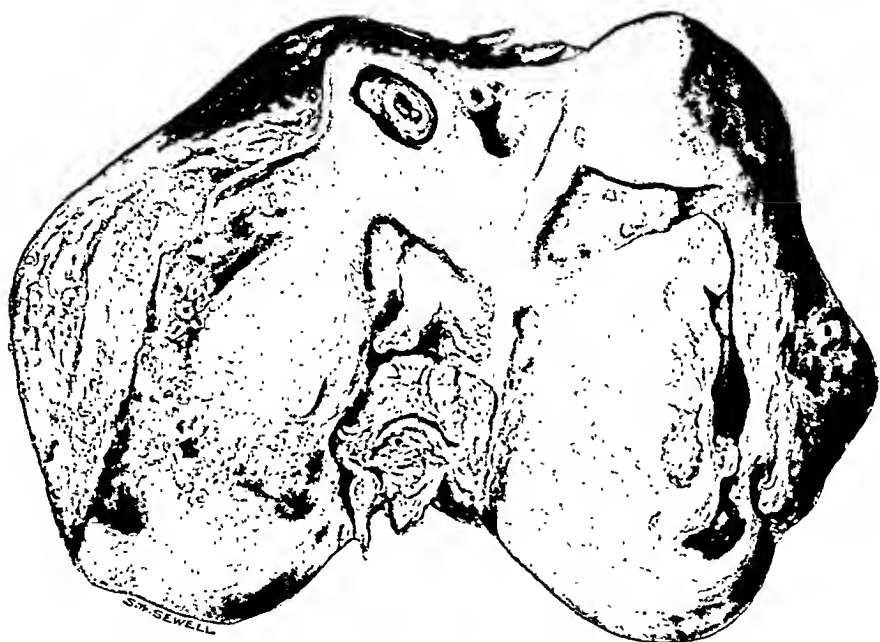
ACUTE SUPPURATIVE ARTHRITIS.

The lower articular extremity of the femur.

The cartilage is eroded as the result of suppuration within the joint, the destruction being greatest over the parts exposed to pressure by the articular surfaces of the tibia and patella. The exposed bone is unaltered except over a small area on the posterior aspect of the external condyle, where there is slight ulceration.

Hunterian Museum, R.C.S. 4498.1

CLINICAL HISTORY.—Suppuration followed penetration of the joint by a rusty nail. The joint was first opened and washed out without benefit. Amputation six weeks after injury.



HUNTERIAN MUSEUM, R.C.S. 4498.1

FIBROUS ANKYLOSIS OF KNEE.

The articular surfaces of the right knee-joint torn apart.

The articular cartilage has been destroyed and replaced by fibrous tissue except over the posterior part of the external condyle of the femur. The underlying bone is exposed in places. The semilunar cartilages have disappeared.

Hunterian Museum, R.C.S. 617.1

CLINICAL HISTORY.—The patient was a woman, aged 32, who suffered from phlebitis of the left leg after the birth of her first child. A mild arthritis of the right knee-joint followed, and the joint gradually became ankylosed. Finally only five degrees of painful movement was possible.



HUNTERIAN MUSEUM, R.C.S. 617.1

HÆMARTHROSIS OF KNEE-JOINT.

The lower end of a femur, together with the patella and adjoining synovial membrane. The patella has been turned to one side.

The articular cartilage shows no change, but the synovial membrane is stained a deep red colour by the absorption of blood from the joint cavity. In parts of the membrane the staining is brown from decomposition of the blood.

Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne 611.1

CLINICAL HISTORY.—The patient was a man, aged 32, who was standing on a table whitewashing the ceiling, when he fell and injured his left knee. He was treated with Scott's dressing, and used crutches for one week. After this he was able to get about during the day, but always suffered severe pain at night. Six months after the accident he was admitted to hospital.

On examination the external condyle of the tibia was expanded by an endosteal tumour which had perforated the knee-joint and had produced a hæmarthrosis. Exploratory operation showed the tumour to be a sarcoma, and the limb was removed by amputation.



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE, 611/1

GONORRHOEAL ARTHRITIS.



The articular surfaces of a femur and tibia from a knee-joint.

The articular cartilages present areas of erosion over each condyle of the femur and over the head of the tibia, the latter being the less affected. Flakes of necrotic cartilage are loosely attached to carious areas on the internal condyle of the femur. The various structures of the joint are hyperæmic.

Hunterian Museum, R.C.S. 754.1

CLINICAL HISTORY.—The parts were excised to relieve intense pain in a case of acute gonorrhœal arthritis.

XXIV. NEUROPATHIC ARTHROPATHY.

(*Charcot's Joint.*)

NEUROPATHIC arthropathy is a painless disorganization of a joint, and is commonly associated with tabes dorsalis or syringomyelia, though sometimes it is seen as a sequel to cerebral and peripheral neural lesions. Charcot's joints are much more common in syringomyelia than in tabes, but, as the former is a rare and the latter a common disease, tabetic arthropathy is the usual form met with in practice. The distribution differs. In tabes the joints of the lower limb, in syringomyelia those of the upper limb, are most often affected. Both varieties present the same pathological characters.

A Charcot joint occasionally mimics osteo-arthritis so closely that a differentiation between the two conditions can only be established by examination of the central nervous system. The more usual varieties of the disease are characterized by a certain extravagance in their morbid anatomy. They present a combination of hypertrophy and atrophy, with predominance of one factor or the other in any given joint. As a rule, atrophy is seen chiefly at the hip and shoulder, hypertrophy in the knee and other joints.

Hypertrophic Variety of Charcot's Joint.—In the hypertrophic variety irregular knobs sprout from the ends of the bones, and masses of bone and calcareous plates are formed in the capsule and synovial membrane. The cartilages and any intra-articular structures disappear. Owing to coincident atrophy of parts of the bones and stretching of the capsule by fluid effusion, the joint becomes movable in unnatural directions.

Atrophic Variety of Charcot's Joint.—In the atrophic variety all the intra-articular structures disappear and the shafts of the bones end in rounded or irregular points projecting into the distended capsule, which becomes so thin and so adherent to surrounding structures that it can with difficulty be identified as a separate layer. The synovial membrane loses its smooth appearance and develops hypertrophied fringes and pedunculated fibro-cartilaginous bodies which project into the cavity of the joint. The amount of fluid effusion is always large and may be striking. It is originally clear and serous, but may sometimes be stained by blood as the result of injury to the unstable joint. Suppuration occasionally follows infection from a perforating ulcer or through the blood-stream.

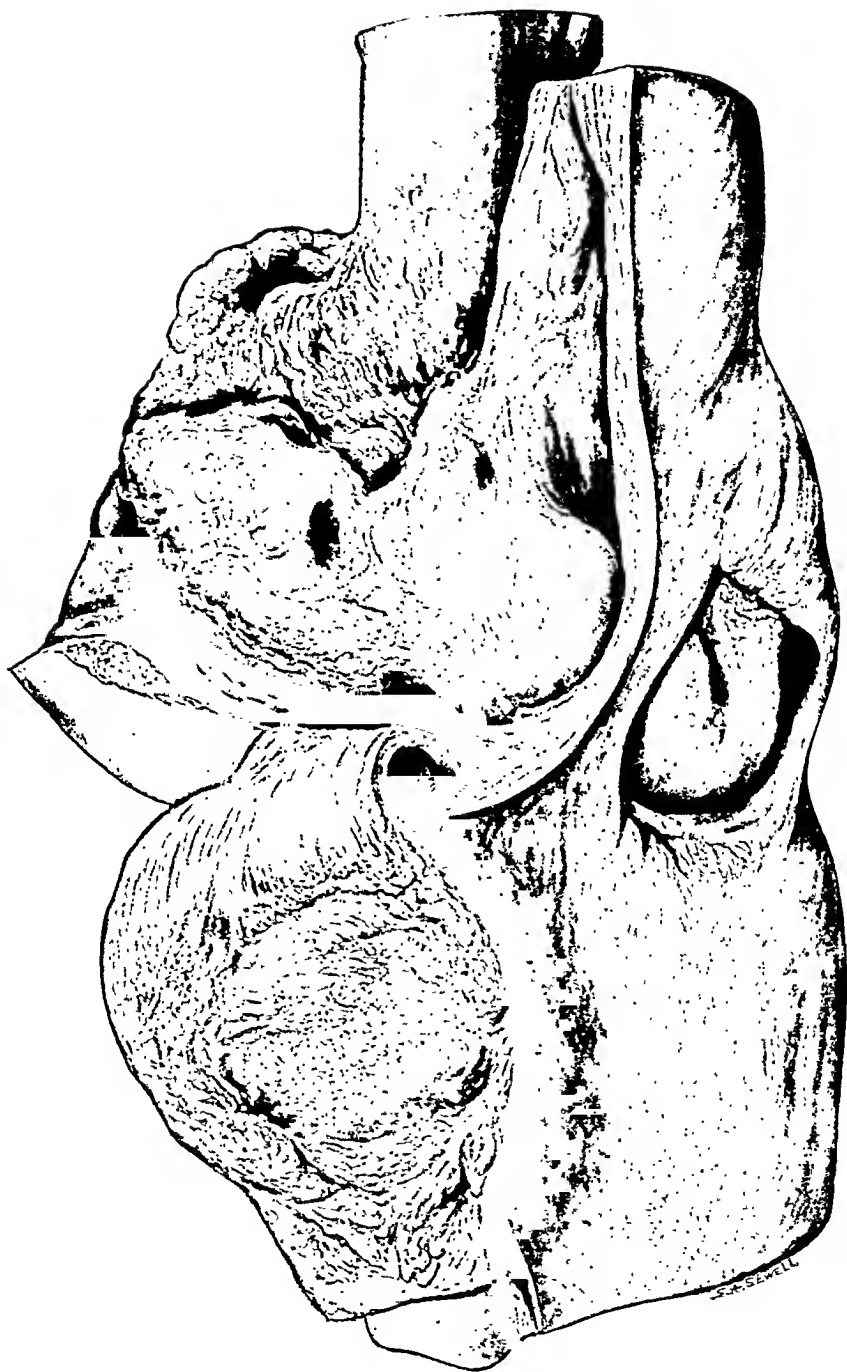
CHARCOT'S DISEASE OF KNEE.

A left knee-joint opened by removal of its anterior wall.

In the skin over the outer side of the knee is a hole through which appears the outer condyle of the femur covered with septic granulations. The articular surfaces are eroded and have large osteophytic masses at their edges. The head of the tibia is deeply excavated.

Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 610/56

CLINICAL HISTORY.—The patient was a diabetic man, aged 43, whose left knee had been crushed by a cart-wheel five years before admission to hospital. His knee was fixed by a Thomas splint, but never fully recovered, and two years later abscesses began to form around the joint. Seven weeks before admission the outer condyle of the femur broke through the skin. The joint was flail-like, and he was unable to walk.



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE, 610/56

CHARCOT'S DISEASE OF KNEE.

A left knee-joint opened from the front.

The capsule is thickened and distended, and the crucial and other ligaments have been destroyed so that the bones can be moved upon one another in any direction.

The head of the tibia is deformed and occupies a deep fossa on the site of the external condyle of the femur. The internal condyle of the femur is intact, but its articular cartilage has largely been replaced by fibrous tissue. Most of the articular cartilage of the tibia and patella has disappeared. The lower end of the femur is almost surrounded by newly-formed bone of irregular shape. The synovial membrane is covered by hypertrophied villi and carries numerous pendulous bodies.

Hunterian Museum, R.C.S., 1023.1

CLINICAL HISTORY.—The patient was a woman, aged 41, who died of general paralysis. There was a strong history of syphilis. Ataxy had been present for eighteen months before the onset of the changes in the joint, and she was ultimately bedridden.



HUNTERIAN MUSEUM, R.C.S., 1023.1

NO. 23—SUPPLEMENT

C 1

CHARCOT'S DISEASE OF KNEE.

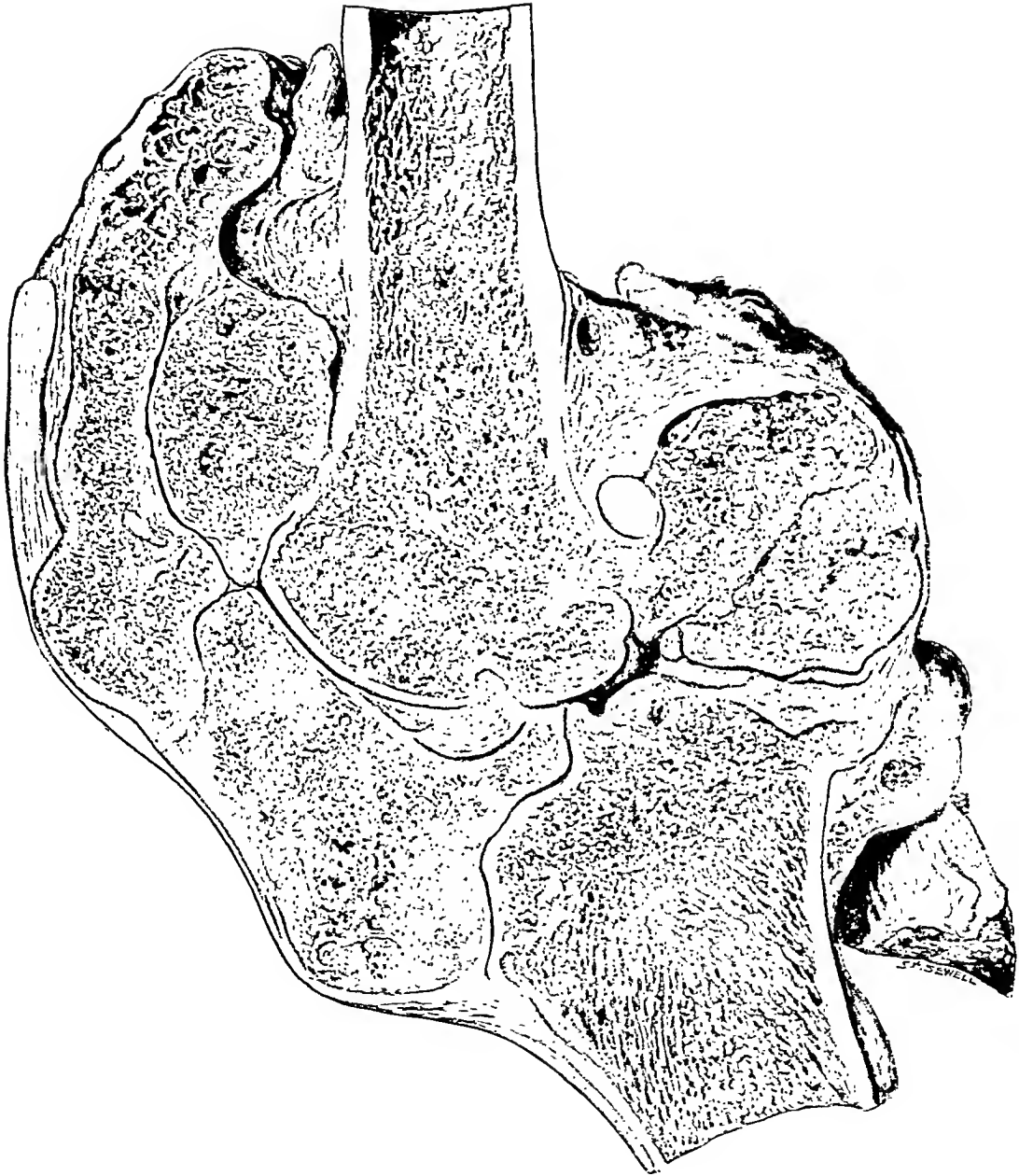
One half of a right knee-joint in sagittal section.

The outlines of the bones forming the articulation are still recognizable, and some articular cartilage remains on the surface of the femur.

Large irregular masses of bone have been formed beneath the synovial membrane, in front and behind the lower end of the femur and above and below the patella. None of these masses are attached to bones except that which extends upwards beneath the quadriceps from the upper border of the patella. A few islands of cartilage are present in the posterior bony mass.

Museum of University College Hospital. 37.B.3

No clinical history.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL. 37.B.3

XXV. ARTHRITIS DEFORMANS.

A RTHRITIS DEFORMANS is a chronic form of joint disease characterized by gradual destruction of the articular surfaces with simultaneous proliferation of certain elements of the joint. The condition is separable into two main types, osteo-arthritis and rheumatoid arthritis. In both, destructive and proliferative changes proceed side by side, and the distinction between them, though largely clinical, is also founded on the presence of bony proliferation in osteo-arthritis, as compared with the predominance of peri-articular fibrosis in the rheumatoid variety. Atrophic changes are common to both forms.

OSTEO-ARTHRITIS.

Osteo-arthritis is characterized by erosion of the working surfaces of the joint, combined with proliferation at the margins of the articular cartilage and on the surface of the synovial membrane. The capsule is not seriously affected.

ETIOLOGY.—Osteo-arthritis affects middle-aged or elderly persons of both sexes, and, as it is a disease of ageing joints, is often regarded as, in part, a degenerative change in articular tissues.

Trauma—particularly in the form of frequently repeated slight injury—is an important cause, and is, indeed, the chief one when the disease commences in the first half of life. Thus, osteo-arthritic changes often accompany repeated internal derangements of the knee in men still of football age. Faulty alignment of the articular surfaces, whether due to fracture into the joint or to deformity, as in knock-knee, is followed by osteo-arthritis with increasing frequency as age advances.

Infective and toxic causes play an important part in the origin and maintenance of osteo-arthritis, in that the disease is often associated with the presence of some focus of chronic inflammation in or around the main apertures of the body. Toxic products absorbed from the alimentary canal in chronic constipation are a possible, though unproved, cause. The influence of cold and damp in increasing the symptoms of the established disease is notorious.

Osteo-arthritis affects chiefly the larger joints, seldom many in a given person, and often only one. When it assumes a polyarticular form it usually commences in the hands and feet, and is then often characterized by the small, bony outgrowths on the phalanges known as Heberden's nodes.

MORBID ANATOMY.—The first signs of osteo-arthritis are atrophic, and appear in the central regions of the articular cartilage, which becomes thin and soft and loses its lustre. Its matrix tends to split along parallel lines running obliquely from the free surface to the bone (fibrillation), and may

develop microscopic cysts. The margin of the articular cartilage is covered by small, dilated vessels extending in for about $\frac{1}{2}$ in. from the edge, and indicating the region in which proliferation will be evident later. The synovial membrane is pink and juicy and secretes a slight excess of synovial fluid. With the atrophy of the central bearing surface of the cartilage, hypertrophic changes appear in those parts of the joint which are less exposed to friction. The margins of the articular cartilage become thickened (lipping), so that they can be felt on clinical examination, and beneath the cartilaginous lip an irregular proliferation of bone marks the site of future osteophytic outgrowth. The villi and fringes of the synovial membrane hypertrophy, and project into the cavity of the joint like miniature strands of pink seaweed. Within their substance fat may be deposited in considerable quantity (lipoma arborescens), or nodules of fibrocartilage may appear and form loose bodies within the joint. In the late stages the synovial membrane may atrophy in association with arteriosclerotic changes in its smaller vessels. Intra-articular structures, such as the semilunar cartilages of the knee, at first develop synovial fringes and later are gradually destroyed. Eventually, the articular cartilage disappears, leaving exposed and in contact with one another bony surfaces which grate on movement.

Atrophy and hypertrophy of the bone-ends follow the corresponding changes in the cartilage, and produce the characteristic picture from which the disease derives its name. The exposed bone becomes worn away into grooves where it is in contact with its fellow, and its surface becomes dense, white, and polished (eburnation). Around the edges of the bearing surfaces projects a ring of osteophytes which represents the proliferative side of this stage of the disease. The combination of destruction of the original shape of the bone-ends with production of osteophytes necessarily leads to loss of mobility, and may end in locking of the joint, quite apart from the loss of function due to pain. Immobility of an osteo-arthritic joint is due to deformation of the articular ends, never to bony ankylosis.

The progress of bony atrophy may be studied in dried specimens from the condition of the head and neck of the femur at successive stages of osteoarthritis of the hip. Erosion of cartilage and lipping are succeeded by wearing away of the upper, weight-bearing surface of the head, and outgrowth of osteophytes in the direction of the neck. When the head of the bone has been destroyed, the process of attrition attacks the upper surface of the neck, and, for a time, the production of osteophytes below keeps pace with destruction of the neck above, so that an apparent coxa vara deformity results. Eventually the whole of the head and neck may disappear. In other cases, erosion of bone is checked by the limitation of movement caused by interlocking osteophytes.

X-RAY APPEARANCES.—Hypertrophic changes are the first to become visible in the bony lipping of the articular edges, which represents the beginning of osteophytic outgrowth. The first evidence of atrophy is the diminution or disappearance of the cartilage gap—the clear space between the ends of the bones occupied by the articular cartilage. In later stages of the disease, deformity of the ends of the bones and osteophytes are the most striking radiographic appearances. Less constant are irregular areas of rarefaction

in the cancellous tissue of the articular extremities, and the small periosteal bony outgrowths on the phalanges known as Heberden's nodes.

Morbus Coxæ Senilis.—Osteo-arthritis of the hip is specially common in elderly persons, and frequently follows injury. Rapid destruction of the head and neck of the femur, with development of osteophytes on the under aspect, produces a coxa vara deformity and great limitation of movement.

Arthritis Deformans of the Spine (*Spondylitis Deformans*).—Osteo-arthritis occurs in the spine, as in other joints, and is characterized by similar changes. In another rare form of arthritis deformans, there are no osteophytes, but the spinal column becomes rigid through ossification of the intervertebral ligaments. Two types are described, one of which affects the spine, hip, and shoulder (Strümpell-Marie type), while the other is limited to the upper dorsal and cervical spine and is associated with localized meningitis (von Bechterew type).

RHEUMATOID ARTHRITIS.

Rheumatoid arthritis is a progressive disease of infective character combining atrophy of the articular surfaces with peri-articular fibrosis. It is polyarticular, usually symmetrical on the two sides, and often begins in the smaller joints of the hands and feet.

ETIOLOGY.—Rheumatoid arthritis affects females three times more often than males, and commences at any time in the first half of life—most commonly between 20 and 40. Its frequent clinical accompaniment of fever, and the constant microscopic evidence of round-celled infiltration in the articular lesions, indicate an infective origin. It is not attributed to any specific organism, but rather to the toxins elaborated in foci of chronic suppuration in some portion of the alimentary, respiratory, or urogenital system.

MORBID ANATOMY.—The local changes commence in the synovial membrane and capsule, which are swollen and infiltrated with round cells. The surface of the synovial membrane is red, and its villi and fringes undergo hypertrophy. From its edges a film of granulation tissue spreads over, softens, and erodes the articular cartilage, the destruction of which is completed by a similar outgrowth of granulation tissue from the underlying bone. Intra-articular structures disappear. Effusion is slight or absent, and the interior of the joint is occupied by hypertrophied synovial fringes in which fat is deposited or fibrocartilaginous nodules formed, just as in osteo-arthritis. Osteophytes are absent.

The more acute phases of rheumatoid arthritis are marked by spasm of the muscles controlling the affected joints. By the fixation thus induced contiguous surfaces of granulation tissue or of exposed bone may be maintained in apposition long enough to allow them to grow together across the articular cavity and produce fibrous or bony ankylosis. The cancellous tissue of the articular ends of the bones becomes rarefied and atrophies where denuded of cartilage and exposed to pressure. Some asymmetry of the joint is thereby produced, but the chief cause of deformity is the contraction which occurs in the capsule. In the early stages of the disease there is a round-cell

infiltration of the peri-articular tissues, which constitutes the pathological basis underlying the clinical sign of fusiform swelling of the joint. Atrophy and contracture of the surrounding muscles contribute to the ultimate deformity and disability, as in the ulnar deviation of the fingers. The overlying skin often shares in the atrophic process and becomes smooth and shiny.

X-RAY APPEARANCES.—Erosion of articular cartilage is shown by disappearance of the cartilage gap between the bones. Atrophy of bone is indicated by diminished density of the cancellous tissue and by alteration in the outline of the articular surface.

Still's Disease.—This is a disease of young children characterized by fusiform swelling of many joints, muscular atrophy, and enlargement of lymphatic glands. The swelling of the joints is due to peri-articular thickening, as in rheumatoid arthritis, but the bones do not atrophy and the cartilages are but slightly eroded. The enlarged lymphatic glands are firm and painless. Their microscopic appearances are those of chronic inflammation, but there is no peri-adenitis and the glands therefore remain discrete. There may be an associated enlargement of the spleen.

OSTEO-ARTHRITIS OF KNEE.

A left knee-joint opened from the front.

The articulating surfaces are rough and studded with raised plaques and nodules of cartilage. Only the lower and posterior part of the external condyle is comparatively smooth as a result of wearing away by movements of the joint. The edges of the femoral condyle are surrounded by lips of cartilage. The upper surface of the external tuberosity of the tibia is almost devoid of articular cartilage. The menisci are ragged and show much chronic inflammatory change. The articular surface of the patella is denuded of cartilage. The synovial membrane of the supra-patellar pouch is thin and smooth, but in other parts of the joint the membrane is roughened by branching villi, some of which are enlarged to such an extent as to form pedunculated tumours. There are two particularly large pedunculated cartilaginous masses arising near the border of the patella.

Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 610/48

CLINICAL HISTORY.—The patient was a woman, aged 55, who died from acute intestinal obstruction. No history of the disease of the knee was obtained.



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE. 610/48

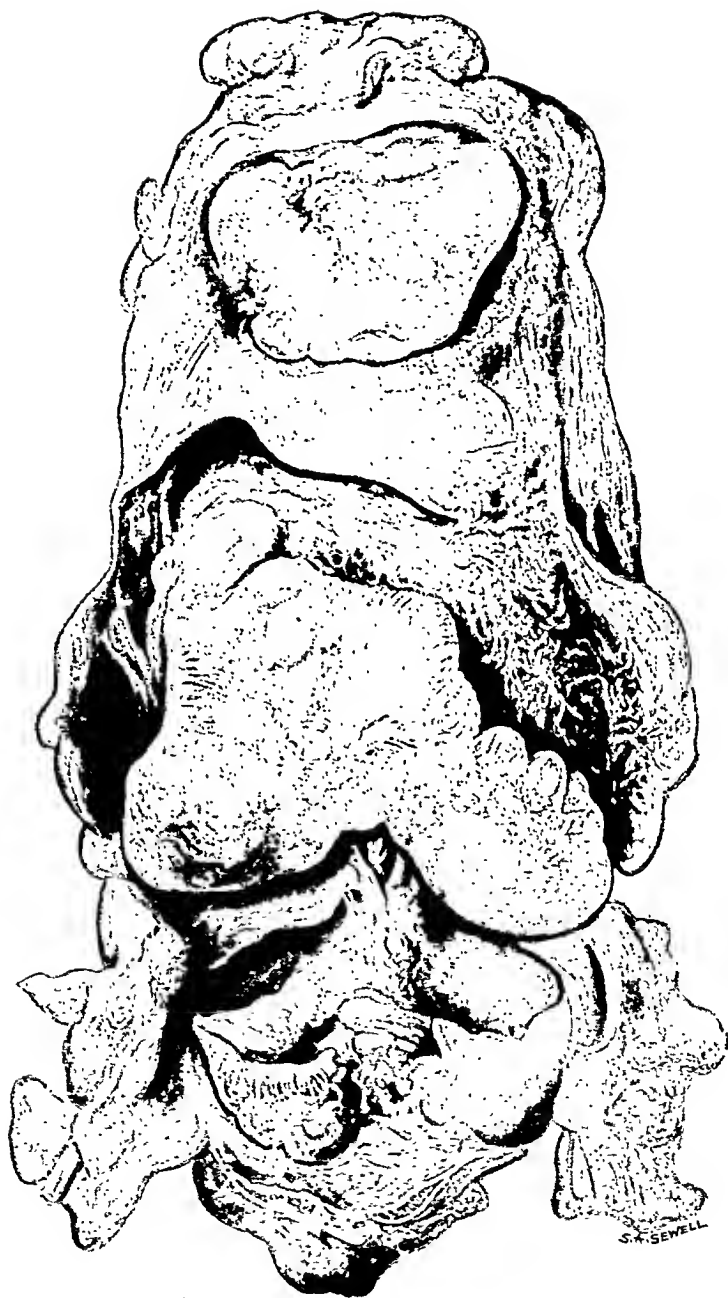
OSTEO-ARTHRITIS OF KNEE.

A knee-joint laid open from the front.

The articular surfaces are irregular from erosion of the centre and new formation of cartilage at the edges. The remaining cartilage is split in a characteristic fashion, and in the centre shows well-marked fibrillation. Over the lower surface of the external condyle of the femur the cartilage is destroyed to such an extent as to expose the underlying compact bone. The edges of the articulating surfaces are lipped, the lipping being supported by underlying osteophytes. The synovial membrane is thick and red, with enlarged villous processes on its surface. The external meniscus is irregular along its free border.

Museum of the College of Medicine, University of Durham, Newcastle-upon-Tyne, 610/49

CLINICAL HISTORY.—The patient was a woman, aged 78, who died from malignant disease of the thyroid gland.



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM, NEWCASTLE-UPON-TYNE. 610/49

OSTEO-ARTHRITIS OF SHOULDER.

The head of a humerus, with a large semilunar piece of the capsule of the shoulder-joint.

The head is denuded of cartilage and surrounded by cartilaginous and osseous outgrowths, and by patches of villous synovial membrane. The compact layer which covered the exposed bone has been destroyed. From the inner surface of the synovial membrane there projects a cluster of pedunculated bodies. The capsule of the joint is thickened.

Hunterian Museum, R.C.S., 4532.1

CLINICAL HISTORY.—The patient was an Army Pensioner whose right shoulder was injured by the tail-board of a lorry, and, four months later, was again struck by the door of a railway truck. After the latter accident the shoulder was black and swollen.

One year after the first accident the joint was explored and chronic inflammatory changes were found. No bacteria grew in the fluid removed from the joint on that occasion. One year later the head of the bone was resected, with the piece of capsule shown.



HUNTERIAN MUSEUM, R.C.S., 4532.1

OSTEO-ARTHRITIS OF HIP.

The dried upper end of the right femur.

The head of the femur has been altered in shape by erosion of bone on its anterior aspect, and by the addition of new bone at the margin of the articular surface.

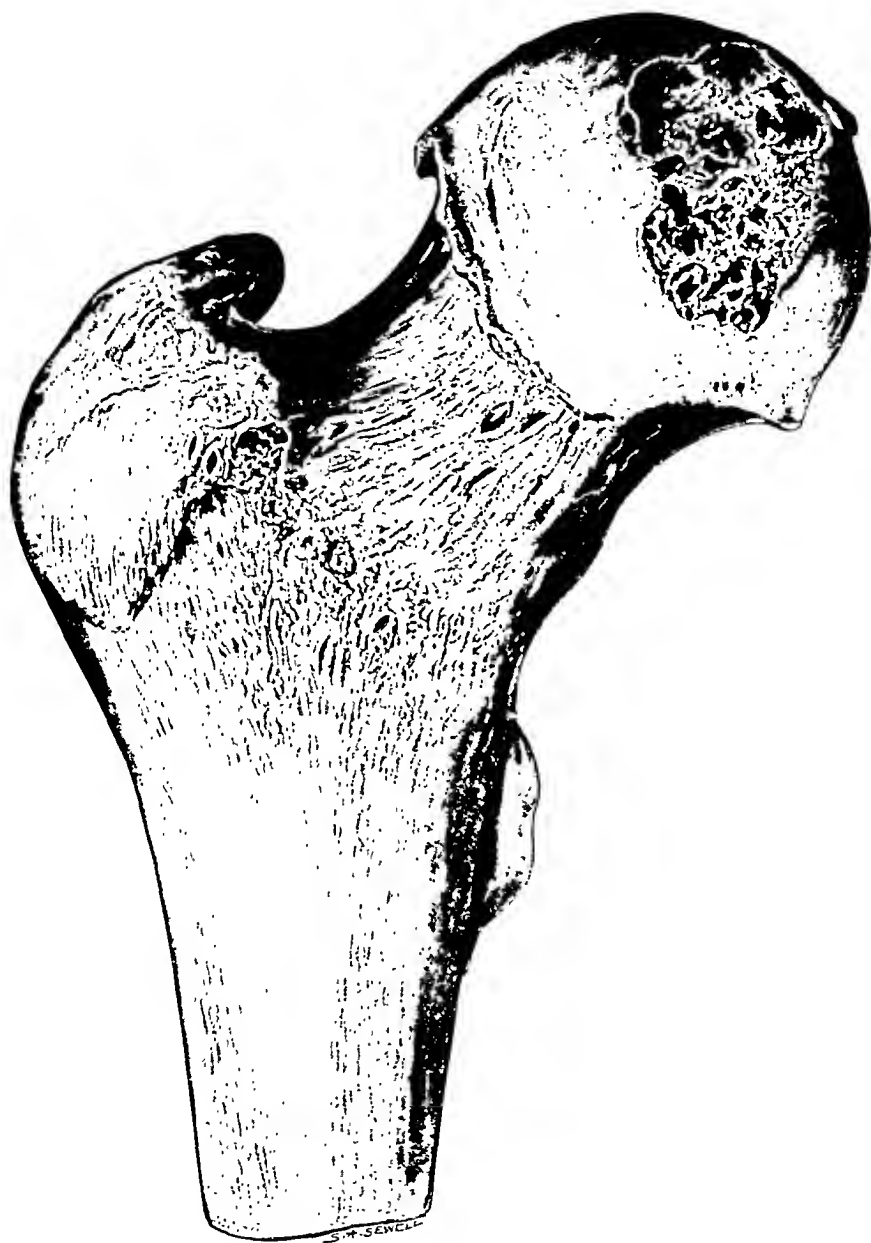
An oval area on the anterior aspect of the articular surface of the head is depressed and uneven. The higher parts of this area between the depressions are dense, white, and highly polished. It is clear that the articular cartilage had disappeared from this eburnated portion of the bone. The rest of the articular surface appears to have been covered with cartilage during life.

Around the articular margin of the head there is a thin rim of new bone which projects out over the adjacent portion of the neck and represents a fused ring of osteophytes.

The neck of the bone is normal.

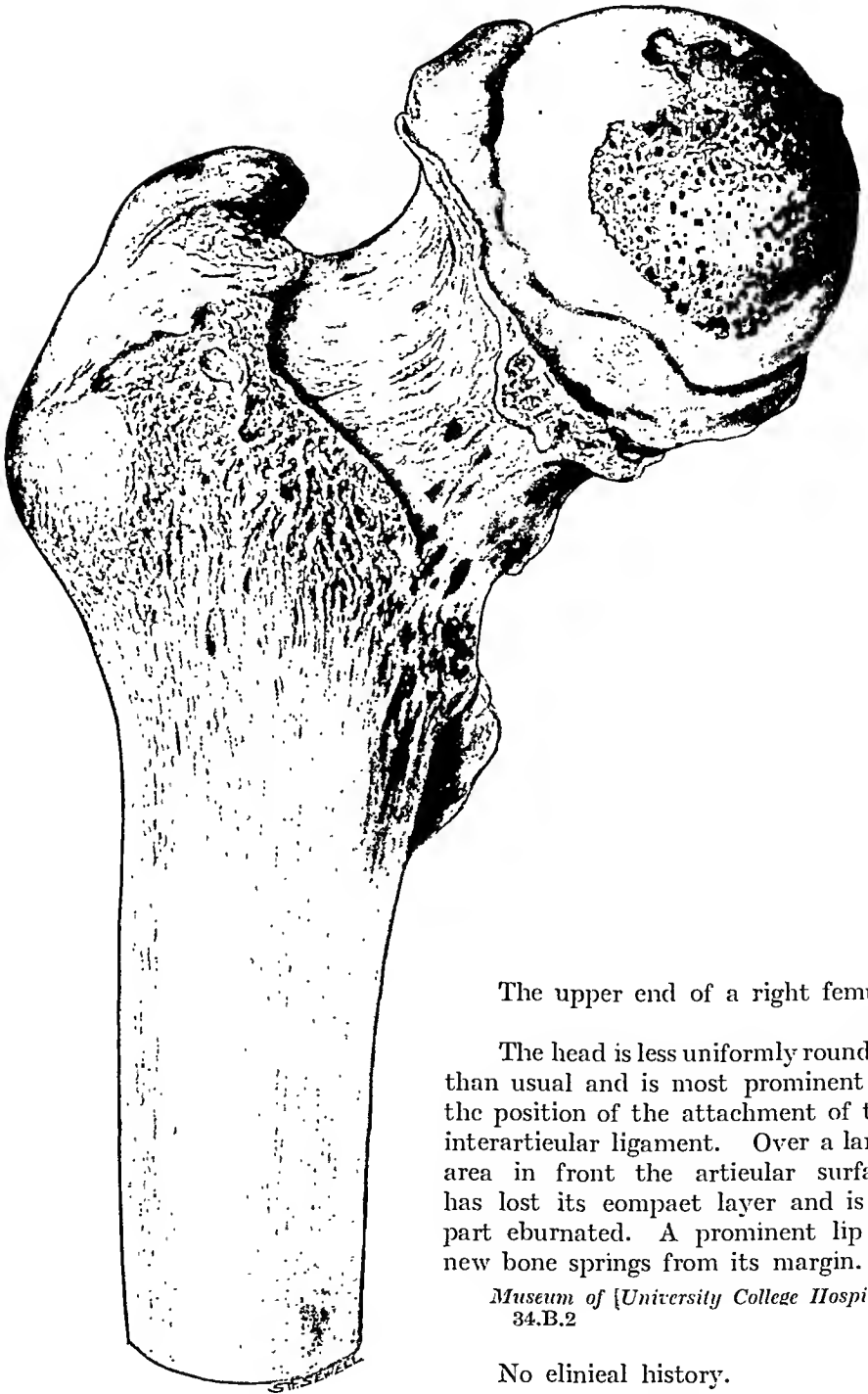
Museum of University College Hospital, 34.B.1

No clinical history.



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 34.B 1

OSTEO-ARTHRITIS OF HIP.



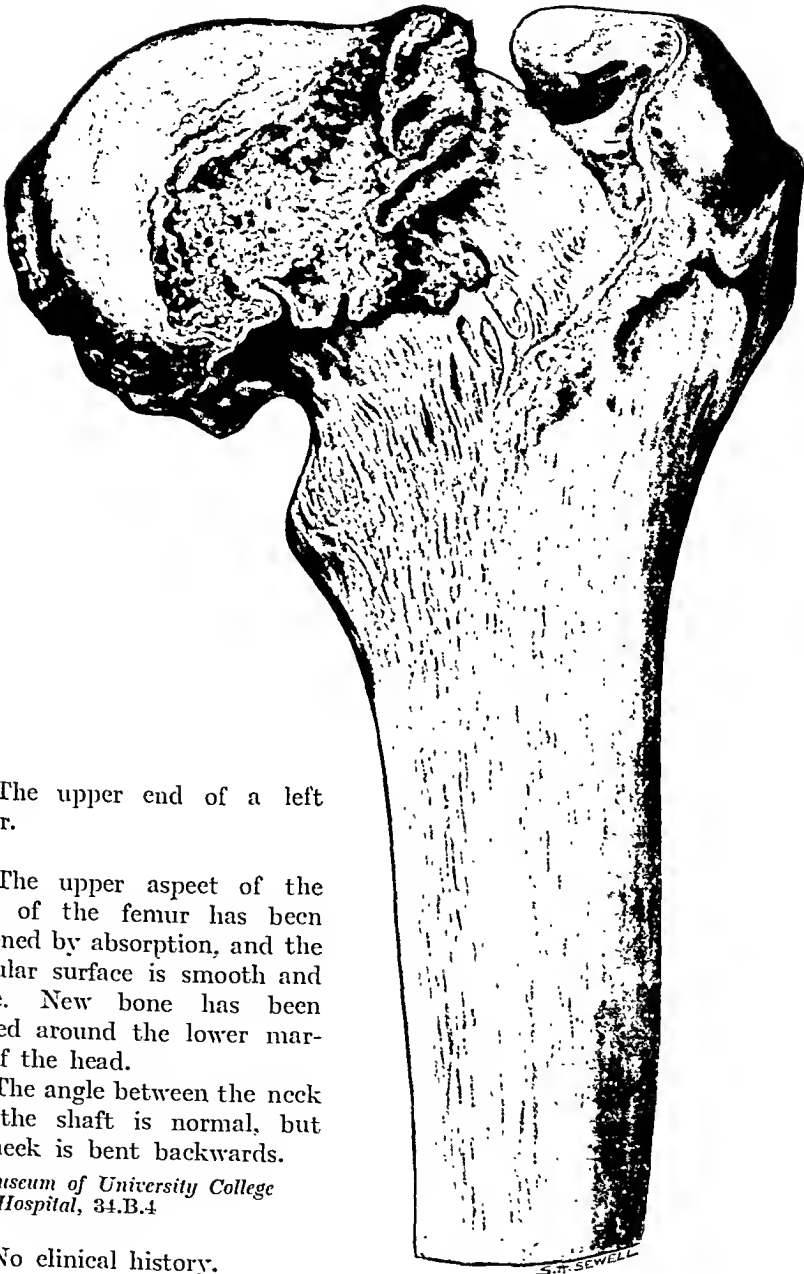
The upper end of a right femur.

The head is less uniformly rounded than usual and is most prominent in the position of the attachment of the interarticular ligament. Over a large area in front the articular surface has lost its compact layer and is in part eburnated. A prominent lip of new bone springs from its margin.

Museum of [University College Hospital,
34.B.2

No clinical history.

OSTEO-ARTHRITIS OF HIP.



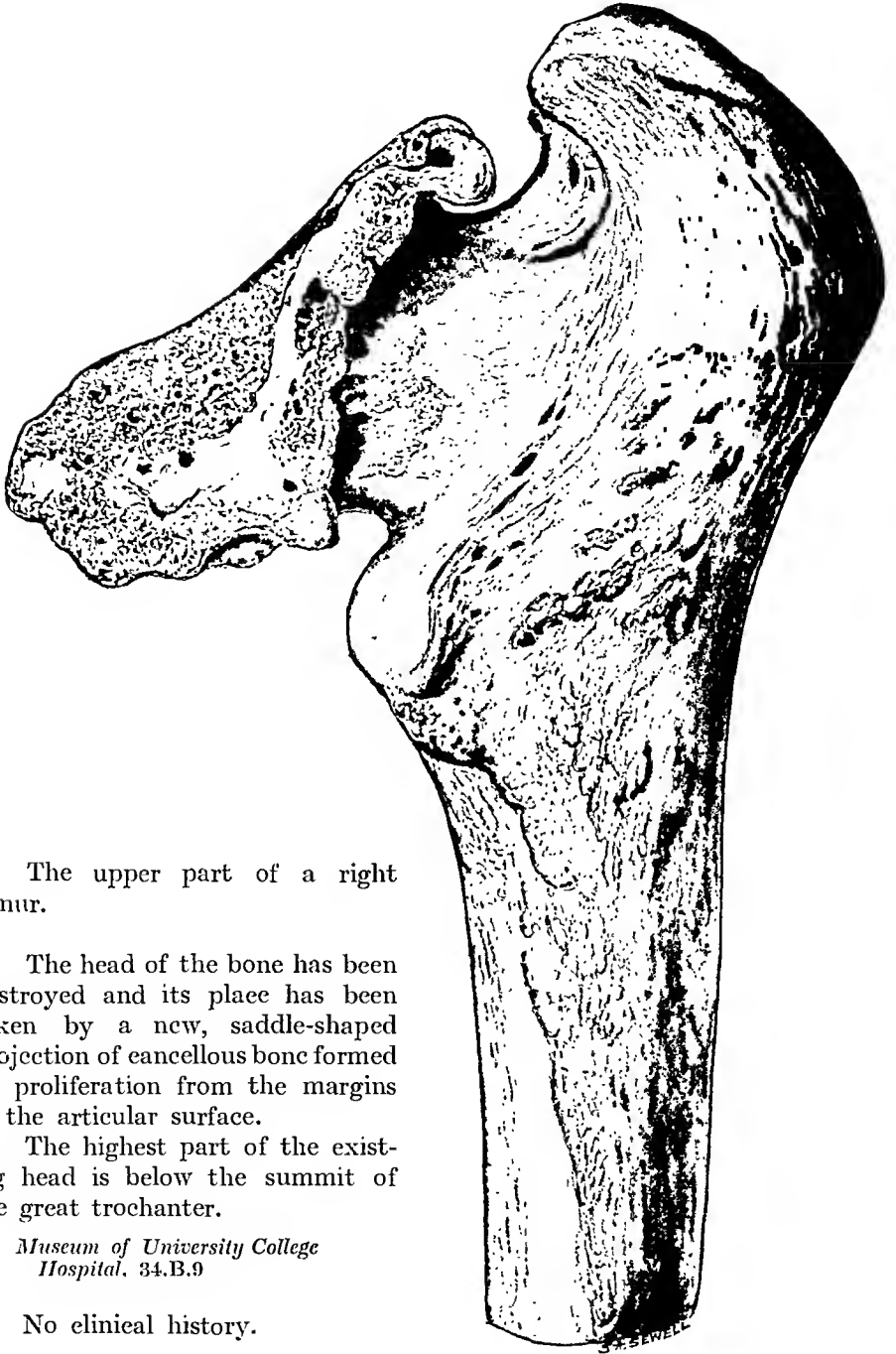
The upper end of a left femur.

The upper aspect of the head of the femur has been flattened by absorption, and the articular surface is smooth and dense. New bone has been formed around the lower margin of the head.

The angle between the neck and the shaft is normal, but the neck is bent backwards.

*Museum of University College
Hospital, 34.B.4*

No clinical history.



The upper part of a right femur.

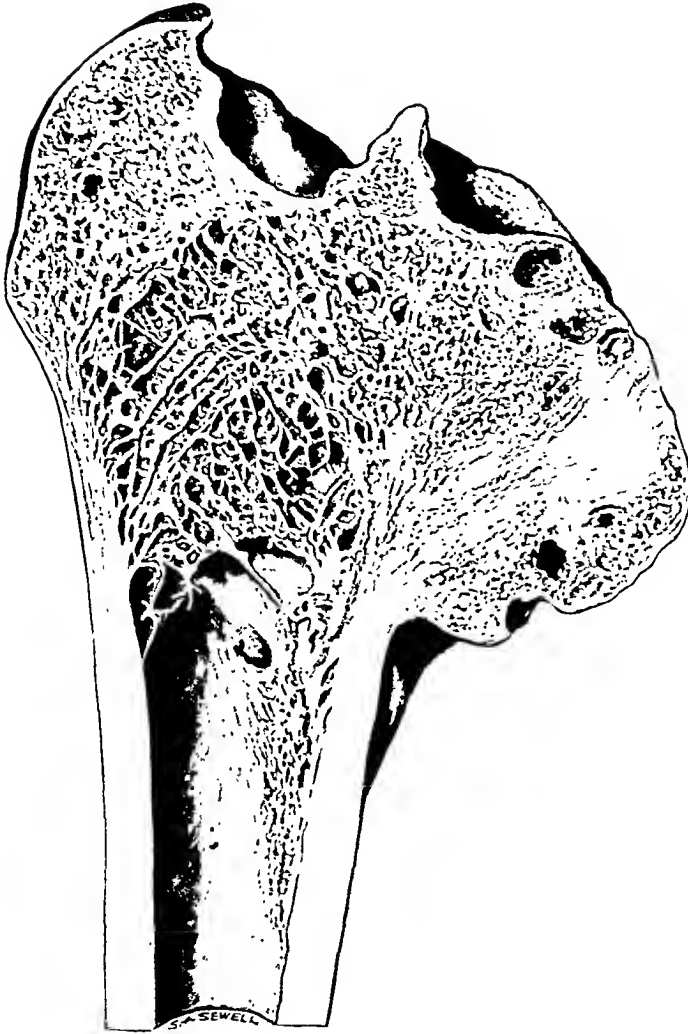
The head of the bone has been destroyed and its place has been taken by a new, saddle-shaped projection of cancellous bone formed by proliferation from the margins of the articular surface.

The highest part of the existing head is below the summit of the great trochanter.

*Museum of University College
Hospital, 34.B.9*

No clinical history.

OSTEO-ARTHRITIS OF HIP.

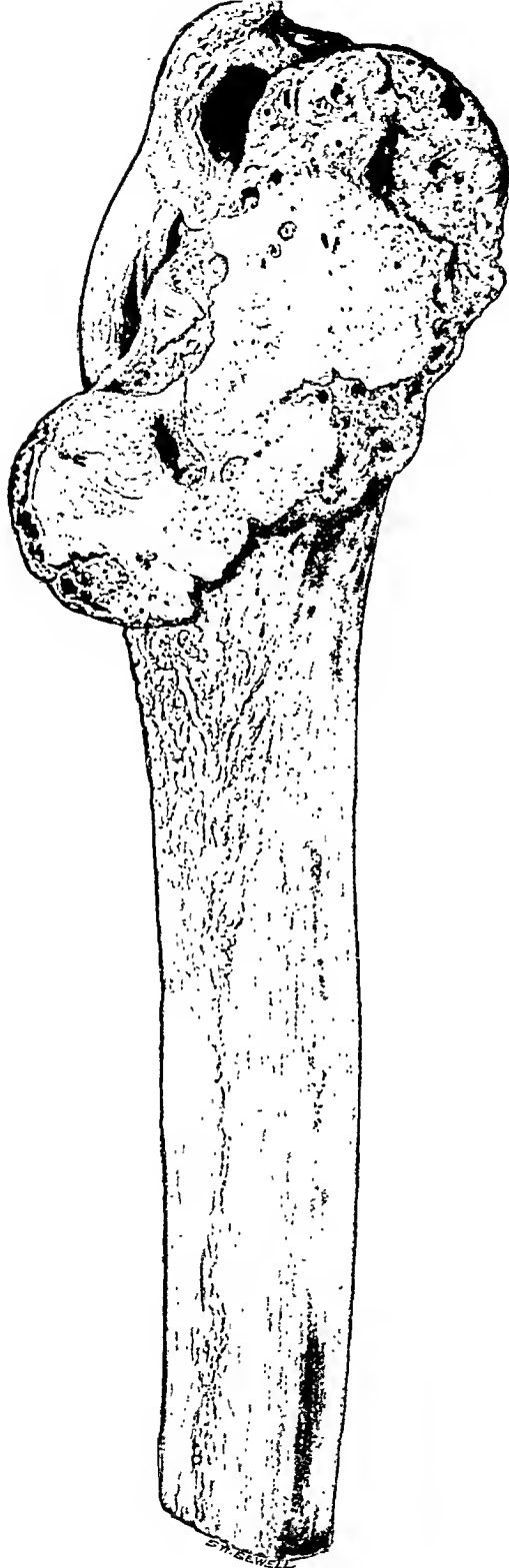


One half of the upper extremity of a left femur divided by coronal section.

The head of the bone has been completely removed by osteo-arthritis, and the articular surface is formed by the remains of the neck and by new bone thrown out around its lower part.

Museum of University College Hospital, 34.B.5

No clinical history.



OSTEO-ARTHRITIS OF HIP.

The upper third of a left femur.

The head, the greater part of the neck, and the upper part of the lesser trochanter have disappeared as the result of absorption in osteo-arthritis.

The elongated articular surface remaining is hard, white, and glazed like poreelain.

*Museum of University College
Hospital, 34.B.6*

No clinical history.

XXVI. CONGENITAL DEFORMITIES OF THE THYROID GLAND.

THE thyroglossal tract from which the great erpart, or more probably the whole, of the thyroid gland is developed, is an outgrowth from the primitive pharynx. It is lined by columnar epithelium and carries a small amount of lymphoid tissue in its wall. The thyroglossal tract passes ventrally to the hyoid arch, with which it acquires a very close connection—so close, indeed, that when it persists into extra-uterine life it may be inseparable from the hyoid bone.

The following pathological conditions may result from persistence of the thyroglossal tract:—

1. **Above the Hyoid.**—A cyst or a mass of thyroid tissue may occupy the position of the foramen cecum on the dorsum of the tongue, where it is exposed to infection or may become the seat of goitrous changes. Such a lingual thyroid may be the only representative of the gland. A cyst containing clear fluid and situated between the two halves of the tongue may be derived from the thyroglossal tract.

2. **Below the Hyoid.**—Persistence of that part of the thyroglossal tract which runs between the hyoid and the isthmus of the thyroid gland is fairly common. The usual form assumed is that of a cyst which lies deep to the infrahyoid muscles, slightly to one side of the middle line. It is lined by columnar or cubical epithelium and its wall contains thyroid vesicles and lymphoid nodules. A thyroglossal cyst may make its first clinical appearance at any age from birth to that of thirty or more years, but seldom reaches a noticeable size until it becomes infected. When infected, it enlarges downwards and eventually bursts through the skin over its lower pole. After discharging its contents the cyst shrinks, and the mouth of the resulting sinus is drawn up in the centre of a number of disfiguring puckers of the skin. The apparent size of a thyroglossal cyst may be increased by an inflammatory fluid distension of the thyrohyoid bursa.

The lower end of the thyroglossal tract is often represented by a pyramidal lobe.

Aberrant portions of thyroid tissue in the line of the thyroglossal tract are liable to the same diseases as the gland itself. They are occasionally found beneath the sternomastoid above and separate from the lateral lobe of the normal thyroid.

THYROGLOSSAL CYST.

The tongue and viscera of the neck drawn from the front.

The thyrohyoid muscles have been separated to expose the thyroglossal cyst, in the anterior wall of which an opening has been cut. The cyst has a thin fibrous wall and a smooth lining. It is attached above to the hyoid, and below to a pyramidal process of the isthmus of the thyroid.

Museum of the University of Manchester, 24.183

No clinical history.



MUSEUM OF THE UNIVERSITY OF MANCHESTER, 24.183

XXVII. INFLAMMATION OF THE THYROID GLAND.

ALL forms of thyroiditis are rare. Acute non-suppurative thyroiditis in a previously healthy gland, or in a diffused goitre, is generally caused by tonsillitis, influenza, acute rheumatism, or measles. It may be followed by disturbance of the metabolic activity and structure of the gland, either in the form of toxic goitre or of myxœdema.

Suppuration occurs chiefly when a goitrous nodule is infected, as it may be in severe septic diseases. The pus is at first confined within the gland, but, if not released, it is apt to burst through the capsule and pass downwards behind the pre-tracheal fascia to the mediastinum, or to perforate the trachea. Subacute suppuration may occur in nodular goitres of long standing during typhoid fever, or without recognizable cause. When a cyst is infected it rapidly increases in size and the surrounding thyroid tissue becomes distended by œdema. This facilitates enucleation of the infected cyst.

CYSTIC ADENOMA OF THYROID.
(SUPPURATION.)



One lateral lobe of a thyroid gland divided by coronal section.

The lateral lobe is distended by an adenomatous mass which is enclosed by a thick fibrous capsule. The compressed upper pole of the gland is still recognizable. The central part of the tumour is solid. Above and below are cysts with a necrotic lining.

Museum of University College Hospital, 11.D.1

MICROSCOPIC STRUCTURE.—Round-celled infiltration of cyst wall.

No clinical history.

XXVIII. GOITRE.

GOITRE is a clinical term meaning enlargement of the thyroid gland. If inflammatory swellings and malignant tumours are excluded, the category of simple goitres comprises hyperplastic and degenerative changes, with which variations in the biological activities of the gland may or may not be associated.

Goitre develops chiefly between the ages of 15 and 30, but may—and in countries where it is endemic frequently does—commence during intra-uterine life. In childhood it is equally common in boys and girls, but from puberty onwards all kinds of goitre are more common in the female than in the male.

Although abnormality of structure in the thyroid is frequently associated with disorder of function, the clinical picture of a given case of goitre cannot be deduced from consideration of the naked-eye and microscopic appearance of the gland, except in typical exophthalmic goitre (Graves' disease) and in some cases of myxœdema or cretinism where thyroid tissue is almost or entirely lacking. The reason for this difficulty in co-ordinating anatomical structure with physiological activity is that, while the appearance of a goitre is largely dependent on the amount of colloid which it contains, the colloid content is not an index of the secretory activity of the gland. The epithelium of the thyroid secretes at least two substances. One of these passes directly into the systemic circulation, either through the wall of the capillaries which surround the acini, or through the lymphatics; the other is stored as colloid within the lumen of the acini and is excreted thence as required by the organism. The quantity of colloid in the gland is therefore a reserve stock carried between secretion and excretion, and is not an index of the rate at which the substance is passing from the thyroid to the rest of the body. The iodine content of the gland is carried mainly in the colloid material, and to a lesser degree in the cells of the secreting epithelium. A goitre does not spring into existence suddenly, but is a gradually progressing disease commencing in the epithelium of the thyroid, and bringing in its train certain secondary changes in the connective-tissue stroma of the gland. Since the storage of colloid is one of the functions of the thyroid, it is natural to find variations in the colloid content of the vesicles in different kinds of goitre, and to find some goitres which contain little or no colloid.

VARIETIES OF SIMPLE GOITRE.

CLASSIFICATION—

Diffuse Goitre.—

1. Enlargement due to multiplication of epithelium without colloid storage :—
 Parenchymatous goitre.
 Exophthalmic goitre (Graves' disease).
2. Enlargement accompanied by excess of colloid :—
 Diffuse colloid goitre.

Nodular Goitre.—

1. Enlargement due to multiplication of epithelium without colloid storage :—
 Parenchymatous adenoma.
2. Enlargement accompanied by excess of colloid :—
 Multinodular colloid goitre.
 Colloid adenoma.

When a previously normal thyroid becomes goitrous the primary change is in the epithelium of the gland, and the histological appearance of the resulting goitre will depend upon whether the vesicles are or are not in the process of storing colloid during its formation. The anatomical structure of goitres shows certain characteristic appearances at different ages.

Diffuse Goitre.—

DIFFUSE GOITRE OF CHILDHOOD.—In childhood, enlargements of the thyroid are diffuse, and the gland retains its normal shape and uniform consistence. The swelling is due to an increase in the number of epithelial vesicles, often combined with a decrease in their size. The structure of the individual vesicles still resembles that of the normal thyroid. At puberty, the simple goitre of childhood is replaced by one which is still of uniform consistence, but which may present either of two types of structure on section.

DIFFUSE PARENCHYMATOUS GOITRE.—In the first form the store of colloid disappears and the epithelium shows signs of increased activity by elongation of its cells into a cylindrical shape, by multiplication of the layers which form the walls of the vesicles, by desquamation, and by proliferation into the lumen in the form of papillae. This type of diffuse goitre is known as parenchymatous, and is the common goitre of puberty. A diffuse enlargement of moderate degree and with normal thyroid structure may also arise at puberty and during pregnancy, and is probably to be regarded as a work hypertrophy in response to the increased demands made upon the thyroid at that period of life.

Exophthalmic goitre, which is structurally a variety of diffuse parenchymatous goitre, is described separately later.

DIFFUSE COLLOID GOITRE.—The second form is characterized by increase in the colloid content of the vesicles, which become irregularly distended without noticeable alteration in their epithelial lining, except for the flattening consequent on distension. The excess of colloid renders the substance of the gland translucent if a thin slice is held up to the light.

The diffuse goitres of childhood and adolescence involve the whole of the thyroid in a uniform enlargement without change of shape. In early adult life increase in size of the thyroid ceases to be uniform and becomes irregular. The goitre, whether parenchymatous or colloid, is then described as nodular.

Nodular Goitre.—In a nodular goitre the irregular overgrowth may be general, and the nodules are then imperfectly separated from one another, as in the common multinodular colloid goitre, or a local overgrowth may be sufficiently encapsuled to justify its description as an adenoma.

MULTINODULAR COLLOID GOITRE.—This is the common goitre of adults. The thyroid is enlarged by an irregular overgrowth of alveoli, accompanied by a storage of colloid in excess of the normal. The gland becomes converted into a number of nodules of varying size, each of which is surrounded by a capsule of condensed fibrous tissue. These capsules are incomplete, and are of the nature of party walls between the nodules, so that it is difficult to detach one nodule from its neighbours. The larger nodules have a fibrous centre which often contains calcareous granules, and from which fibrous strands radiate outwards towards the periphery.

The quantity of colloid varies within wide limits, but is usually sufficient to impart a translucent appearance to a thin slice of the goitre. Colloid cysts are fairly common.

ADENOMA.—An adenoma of the thyroid is not a simple tumour growing in an otherwise normal gland. The rest of the gland is always goitrous, however much it may be compressed by the pressure of the growing adenoma. This is seen most clearly in the multinodular colloid goitre, where the individual nodules are imperfectly separated from one another; but even in the most completely encapsuled, single, parenchymatous adenoma the rest of the gland is in the state of parenchymatous goitre. The capsule of an adenoma is a fibrous layer derived from the connective-tissue stroma of the gland. Since adenoma of the thyroid is only a special development of nodular goitre, it presents the same two types of structure, parenchymatous and colloid.

The *Parenchymatous Adenoma* is a soft, opaque, white, fleshy tumour, containing no colloid, and composed of irregular alveoli of spheroidal epithelium separated from one another by a vascular connective tissue. It bears a general resemblance to the early foetal thyroid, and is sometimes, on that account, described as a foetal adenoma. Another variety is composed of tubules and vesicles lined by cylindrical epithelium from which project papillæ, as in the parenchymatous goitre of adolescents and in exophthalmic goitre.

The *Colloid Adenoma* is firmer than the parenchymatous adenoma, and its cut surface has a translucent appearance owing to the quantity of colloid present. At its centre there is usually a mass of fibrous tissue, containing calcareous granules, from which incomplete fibrous septa radiate towards the capsule. The microscopic structure is similar to that of diffuse colloid goitre. Cystic degeneration is common in colloid adenomata, especially in those of large size.

SECONDARY CHANGES IN GOITRE.

Cystic Degeneration.—Cysts are common in nodular colloid goitres. Colloid cysts are formed by absorption of the septa between neighbouring vesicles which are excessively distended by colloid. They are also formed by mucoid degeneration of the interstitial tissue of the nodules. Every cyst is surrounded by a fibrous capsule, which varies in thickness, and is formed by condensation of the surrounding interstitial tissue of the gland. The capsule can be separated without difficulty from the compressed thyroid tissue which surrounds it. The lining of the cyst may be smooth, or may be roughened by degenerating thyroid tissue. The contents of the cyst are either colloid or serous fluid, which, in turn, may be clear or may contain cholesterol crystals, or be coloured by the products of hæmorrhage.

Fibrosis.—Fibrosis is common, though to a variable degree, in both diffuse and nodular goitre. Reference has already been made to the fibrous centre of a colloid nodule, in addition to which a fine, diffuse fibrosis may occur throughout a part or the whole of a goitre. On section the part affected is paler than the rest of the gland.

Fibrous Goitre.—Excessive fibrosis in a diffuse goitre may give the gland a uniformly tough consistence, and, on section, produce an appearance of small islets of thyroid tissue embedded in a mass of fibrous tissue the shape of the gland.

Calcification.—In nodular goitres the fibrous centres of the nodules are often calcified. Calcification is also common in the walls of cysts. A

calcified cyst forms a stony-hard but movable mass, the shadow of which shows clearly in a radiograph.

Hæmorrhage.—When hæmorrhage occurs, as it frequently does, into a solid or cystic adenomatous nodule, there is a sudden rise of pressure within the gland and a local increase in size and hardness of the goitre, which compresses the trachea and produces urgent, and sometimes fatal, respiratory obstruction. Within a few days the serous component of the effused blood is absorbed and the tension is correspondingly reduced. Blood may also escape from within the fascial sheath of the thyroid and stain the skin of the neck.

PRESSURE EFFECTS OF GOITRE.

Goitres of all kinds tend by their continued growth to produce mechanical effects on the viscera of the neck. The most consistent of these effects is deviation and compression of the trachea. The trachea is pushed to one side by any asymmetrical enlargement of the thyroid, and is usually compressed at the same time. Bilateral compression without deviation is produced by large, diffuse goitres. Hæmorrhage into a solid or cystic adenoma immediately intensifies the pressure and may even cause sudden death from asphyxia. The degree to which the trachea is affected by a goitre can readily be determined by radiography, a method of special value when the whole or a part of the goitre is within the thorax.

Very large goitres impede the circulation through the great veins of the neck or superior mediastinum and so cause congestion of the face and dilatation of the superficial veins of the neck and upper part of the chest. The recurrent laryngeal nerve may be partially or completely paralysed by the pressure of a simple goitre. The œsophagus is rarely obstructed on account of its mobility.

Intrathoracic Goitre.—In the early embryo the developing thyroid rests upon the pericardium, from which it becomes separated later by the forward growth of the head and neck. There remains always an easy path behind the pretracheal muscles along which a goitre can extend in the loose cellular tissue of the superior mediastinum towards the aorta. In the common form of intrathoracic goitre a nodular colloid goitre sends a narrow prolongation downwards through the upper aperture of the thorax to expand again in the superior mediastinum, or a large goitre extends down from the neck into the thorax and is grooved on its anterior surface by the upper border of the manubrium. In each case the intrathoracic part is situated in front of the great vessels. Much less common is a goitre which grows by a narrow stalk from the posterior surface of one lateral lobe and passes down behind the great vessels into the thorax. A goitre taking this path keeps close to the vertebral column between the œsophagus and the mediastinal pleura. On account of the rigid walls of the cavity on which it encroaches, an intrathoracic goitre is apt to produce serious pressure upon the trachea, the recurrent laryngeal nerve, and the main veins of the neck. It is in intrathoracic goitre that interference with the venous return from the neck is most intense, and that the collateral circulation in the superficial veins at the junction of neck and trunk reaches its highest development. Intrathoracic goitres are liable to the same secondary changes—hæmorrhage, calcification, etc.—as are ordinary cervical goitres.

PATHOLOGICAL CONDITIONS ASSOCIATED WITH DISTURBANCE OF THYROID METABOLISM.

Toxic Goitre.—

EXOPHTHALMIC GOITRE (*Graves' Disease; Primary Toxic Goitre*).—Exophthalmic goitre is the only variety of goitre in which there is a constant relation between the anatomical structure of the thyroid and the clinical picture of the disease. It is much more common in women than in men, and its active manifestations are practically confined to the period of sexual activity.

During life the most striking feature of the thyroid of Graves' disease is the intensity of its arterial blood-supply. The gland itself is uniformly enlarged to a moderate degree, and soft in the early stages, becoming firmer as the disease progresses. On section of the gland after removal, the surface is uniformly pale, opaque, and fleshy, without cysts or visible colloid. The microscopic appearance is that of a parenchymatous goitre with alveoli of moderate size, lined by one or more layers of a low, columnar epithelium, which often forms papillary projections into the lumen. Colloid is absent, or, if present, is in small amount, vacuolated, and difficult to stain. The iodine content of the gland is high. Aggregations of lymphocytes, such as are seen occasionally in non-toxic goitres, are common. The thymus is regularly enlarged in association with the thyroid in exophthalmic goitre, and occasionally a pituitary enlargement with symptoms of acromegaly is observed. The natural termination of surviving cases of Graves' disease is exhaustion of the over-active thyroid. The gland undergoes fibrosis, and symptoms of myxœdema are superimposed on those of hyperthyroidism.

TOXIC ADENOMA (*Secondary Toxic Goitre*).—The clinical signs of hyperthyroidism may be superimposed upon any of the ordinary forms of adenomatous goitre. The toxic effects are, on the whole, less severe than those of typical Graves' disease, and are less likely to include exophthalmos. The association of hyperthyroidism with diffuse colloid goitre is much less common. All forms of toxic goitre tend to show histological evidence of greater epithelial activity than do non-toxic goitres of similar structure, the layers of cells lining the alveoli being increased in number and the individual cells approximating to the columnar shape.

Cretinism.—The condition of cretinism is caused by deficiency of thyroid secretion in early life. The deficiency may be brought about by atrophy of the gland or by the development of a goitre. When the thyroid is atrophied it is represented by some fibrous tissue, in which a few glandular tubules usually survive. The goitre of a cretin is diffuse in childhood, but becomes nodular and often cystic in the course of time. The glandular epithelium is reduced in quantity, but is not necessarily absent.

Myxœdema.—The symptoms of this disease are caused by deprivation of certain elements of the thyroid secretion in adult life. The gland is shrunken, and its epithelium is replaced, wholly or in part, by fibrous tissue. The fibrosis follows an attack of thyroiditis, or results from the exhaustion of long-continued Graves' disease.

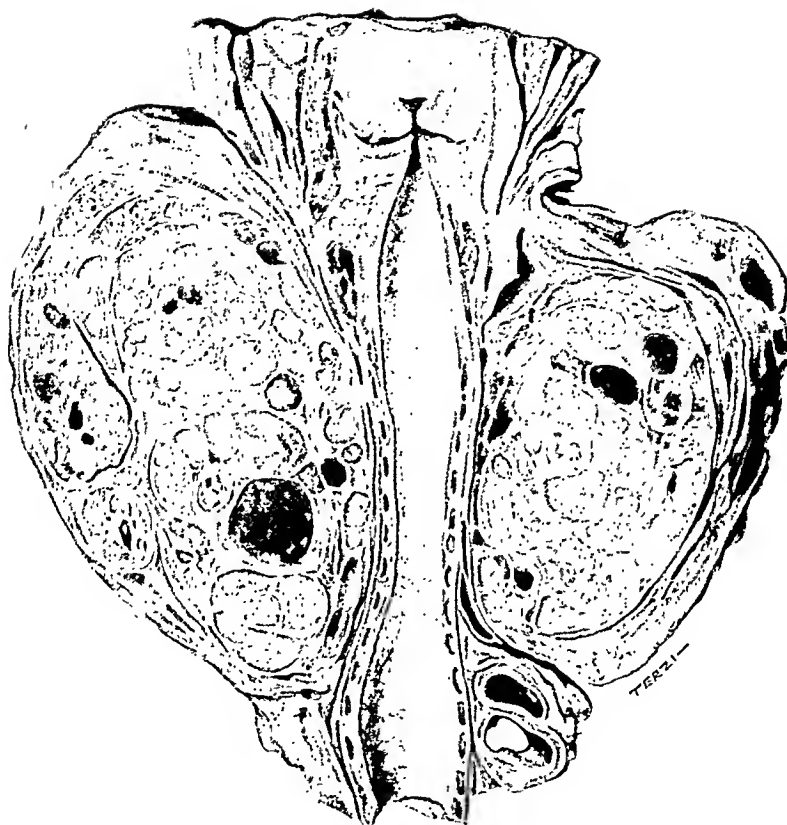
MULTINODULAR COLLOID GOITRE.

One half of a frontal section through an enlarged thyroid gland and trachea.

The greater part of the thyroid is converted into a mass of cysts varying in size up to half an inch in diameter. The cysts are distended with colloid, which, in some places, is mixed with blood and contains small deposits of calcareous matter. The trachea is compressed laterally to less than one half of its normal width.

Hunterian Museum, R.C.S., 7156.1

CLINICAL HISTORY.—The goitre had existed for many years and had caused increasing dyspnoea. The patient died of pneumonia.



HUNTERIAN MUSEUM, R.C.S., 7156.1



HUNTERIAN MUSEUM, R.C.S., 7156.1

× 100

MICROSCOPIC STRUCTURE.—The goitre is composed of thyroid vesicles of irregular size and shape, lined by cubical or flattened epithelium.

COLLOID ADENOMA OF THYROID.

An adenoma of the thyroid gland, bisected.

The tumour is 8 cm. in its longest diameter ; its surface is smooth and covered in all parts by a fibrous capsule. The cut surface presents a dense, bluish-white, fibrous, central portion, surrounded by a brown translucent tissue composed of a delicate meshwork containing colloid substance. Small recent hæmorrhages are present in the tumour.

Museum of University College Hospital, 8.D.1

MICROSCOPIC STRUCTURE.—There is an abundant finely fibrillated stroma, in which are vesicles containing colloid substance mixed with altered blood. Small buds of epithelial cells extend from the vesicles into the surrounding stroma.

CLINICAL HISTORY.—The patient was a woman, aged 39, in whom a tumour of the neck had been noticed for four years. For two years there had been dyspnœa, and for one year loss of voice. The tumour was situated in the right lobe of the thyroid gland ; the left lobe was slightly enlarged. The trachea was displaced to the left of the middle line ; there was slight stridor with complete aphonia. The patient was very neurotic, the breathing was difficult. The tumour was removed by enucleation. The aphonia persisted after the operation.



A. H. MAXWELL.

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 8.D.1

COLLOID GOITRE IN ACCESSORY THYROID.



An oval mass of thyroid tissue three inches in vertical diameter.

Hunterian Museum, R.C.S., 7161.1

CLINICAL HISTORY.—The patient was a woman who, to her knowledge, had had a swelling on the right side of the neck for nine years. Her mother stated there had been a lump the size of an acorn since infancy. There were no symptoms.

On examination the swelling lay deep to the right sternomastoid near the angle of the jaw. At operation it was found to have no connection with the thyroid gland, which was normal. The upper part of the swelling was grooved by the XIth cranial nerve.

CYSTIC ADENOMA OF THYROID.



Part of one lateral lobe of the thyroid gland.

The cut surface of the upper part shows a slight degree of nodular colloid change. The lower part consists of a cyst with a thick fibrous wall, some of which has been cut away. The interior of the cyst is mostly roughened by disintegrating fragments of thyroid tissue. Here and there it is smooth and glistening with crystals of cholesterol.

Hunterian Museum, R.C.S., 7206.1

MICROSCOPIC STRUCTURE.—The irregular projections into the cyst are composed of thyroid vesicles.

CLINICAL HISTORY.—The patient was a man who had had a swelling of the neck for four years. The swelling at first increased rapidly in size and then became stationary. For six weeks before operation it had increased again, and ultimately reached from the thyroid cartilage to the sternum.

‘CYSTIC ADENOMA OF THYROID.



One half of an oval swelling removed from the thyroid gland.

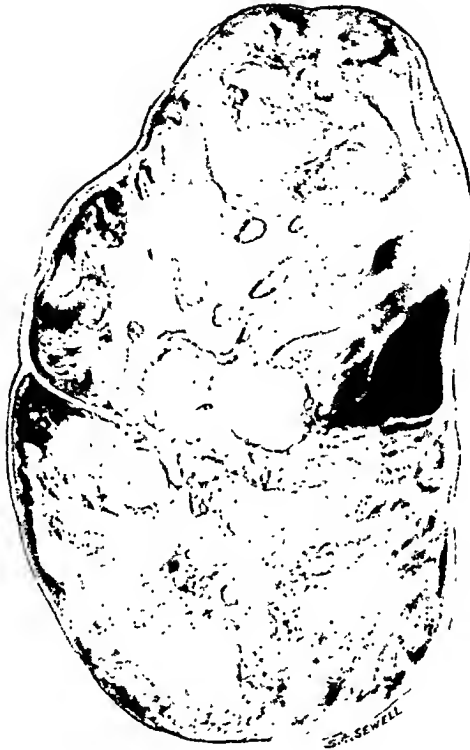
The upper pole is coated with a thin layer of tissue which has the microscopic structure of the thyroid gland. Beneath this is the fibrous wall of the cyst, which contains a translucent coagulum with white, opaque areas of calcification. The blood was probably extravasated at operation.

Hunterian Museum, R.C.S., 7213.1

MICROSCOPIC STRUCTURE.—The cyst wall is composed of dense, hyaline, fibrous tissue within which is a layer of vesicular thyroid.

CLINICAL HISTORY.—The patient was a woman, aged 22, who had noticed a swelling of her neck for two months. At operation it was found to be connected with the isthmus and right lobe of the thyroid.

COLLOID ADENOMA OF THYROID.



One half of an adenoma of the thyroid.

The specimen is roughly oval in outline, nodular on the surface, and surrounded by a fibrous capsule. The centre is occupied by a calcified mass of fibrous tissue. The rest is made up of a fibrous framework in which are enclosed islands of thyroid tissue containing abundant colloid material.

Museum of the University of Sheffield, A.578

MICROSCOPIC STRUCTURE.—Colloid goitre with abundant fibrous stroma.

No clinical history.

CYSTIC ADENOMA OF THYROID.

(HÆMORRHAGE.)



A slice from one lateral lobe of the thyroid gland.

The gland is enlarged by a cyst which has a fibrous capsule, and is surrounded by a layer of compressed thyroid tissue. The cyst is full of clotted blood.

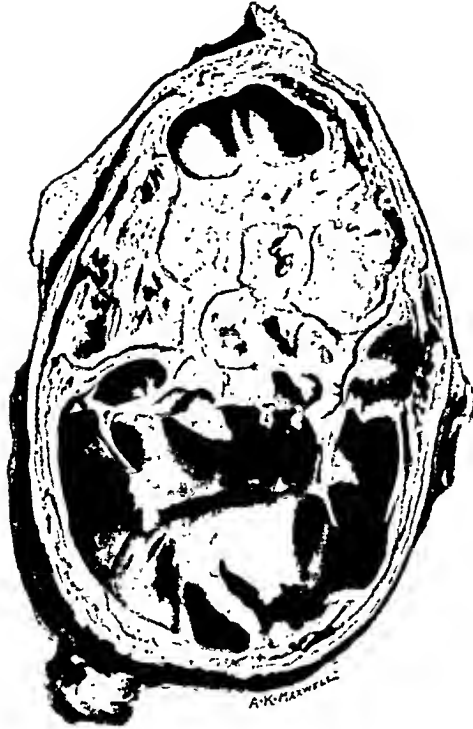
Museum of University College Hospital, 10.D.2

MICROSCOPIC STRUCTURE.—The wall of the cyst shows irregularity of size, contour, and colloid content of the alveoli.

CLINICAL HISTORY.—The patient was a man, aged 51, who had had no symptoms till twelve days before admission to hospital. At intervals of a few days he then had three attacks of pain on the right side of the neck. The first made his collar tight, the second made swallowing difficult, and the third left his voice husky.

On examination there was a rounded, elastic swelling in the right lobe of the thyroid. The right vocal cord was paralysed. Hemithyroidectomy.

CYSTIC ADENOMA OF THYROID.
(CALCIFICATION.)



Part of one lateral lobe of the thyroid gland.

The lobe is distended by a calcified cystic tumour which is bounded by an indistinct fibrous capsule. Outside this is a thin layer of compressed thyroid tissue.

Museum of University College Hospital, 12.D.2

MICROSCOPIC STRUCTURE.—Small vesicles filled with colloid at periphery. Degenerate, structureless, calcified tissue in centre.

CLINICAL HISTORY.—The patient was a man, aged 55, who had a stony hard swelling behind the lower end of the right sternomastoid muscle and sternoclavicular joint. The lump moved on swallowing, and was not attached to surrounding structures. It was opaque to X rays. There were no symptoms.

The swelling was removed by operation, with the upper part of the right lobe of the thyroid. The lower pole was left intact. Convalescence was uneventful.

EXOPHTHALMIC GOITRE.

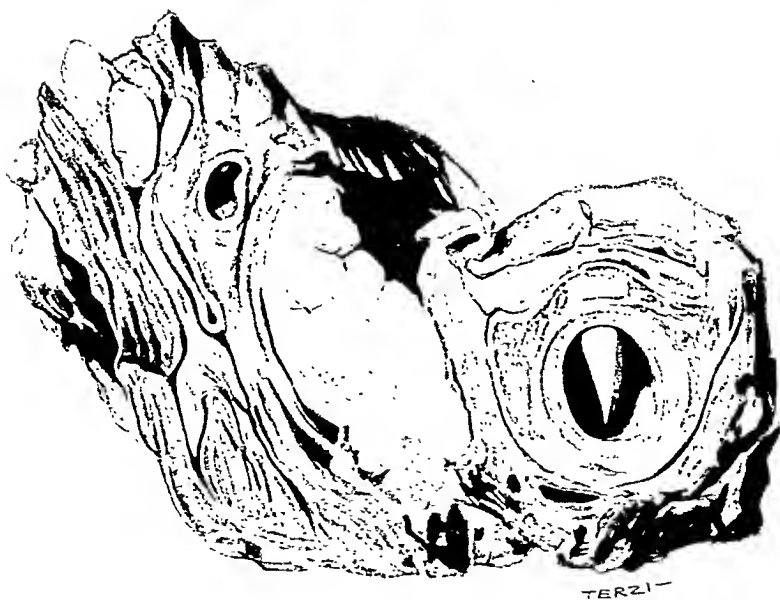
A horizontal section through the larynx and soft parts of the left side of the neck seen from below.

The lateral lobe of the thyroid gland is moderately enlarged, solid, and pale. Blood has been extravasated into the surrounding tissues as a result of the operation for removal of the isthmus and right lobe. The lymph-glands on the postero-lateral aspect of the internal jugular vein are enlarged.

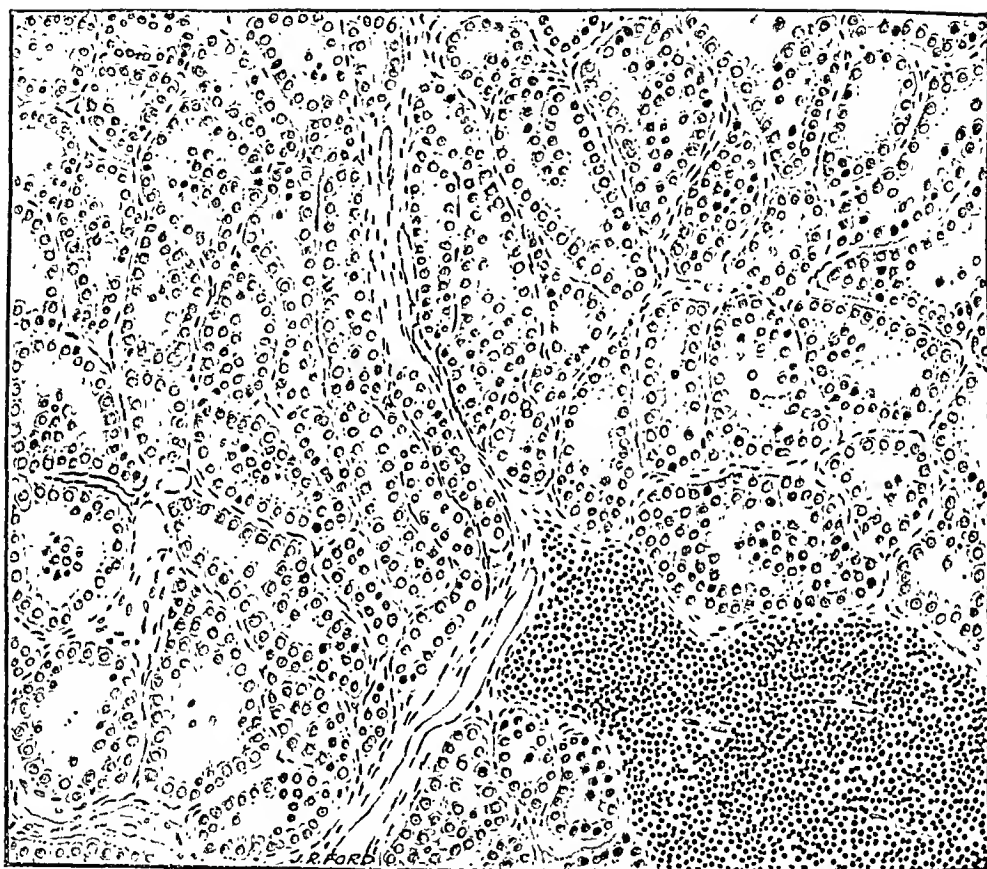
With this specimen is shown a section through the aortic arch and thymus gland from the same patient. The thymus is enlarged, and measures $1 \times 1\frac{1}{2}$ inches. Its left lobe is fleshy, but its right lobe has been largely transformed into fat. There are two lymphoid nodules at its centre.

Hunterian Museum, R.C.S., 7181.1

CLINICAL HISTORY.—The patient had been ill for three months with exophthalmos, tremor, enlargement of thyroid, dyspnœa, dysphagia, and palpitations. Death followed infection of the operation wound.



HUNTERIAN MUSEUM, R.C.S., 181.1



HUNTERIAN MUSEUM, R.C.S. 7181.1.

× 200

MICROSCOPIC STRUCTURE.—The thyroid vesicles are lined by columnar cells and contain no colloid. In the lower right corner is a lymphoid nodule. The blood-vessels are numerous.

